TRANSACTIONS

OF THE

Association of Life Insurance Medical Directors of America

FIFTY-SECOND ANNUAL MEETING

Harry E. Ungerleider, M. D.
Editor

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Transactions of the Association of Life Insurance Medical Directors of America

FIFTY-SECOND ANNUAL MEETING

The Fifty-Second Annual Meeting of The Association of Life Insurance Medical Directors of America was held at the Hotel Pennsylvania in New York City on Thursday and Friday, October 23 and 24, 1941.

PRESIDENT CRAGIN—In opening the fifty-second annual meeting of this Association may I first express my extreme regret that we were unable to hold the meeting in Hartford. However, my home town is in the process of extreme industrial expansion just at present and though we could have fed and entertained you, the question of housing proved entirely inadequate; therefore, New York was selected as next best.

We have tried to have the educational program make up for the lack of golf and other sports; while so far as the entertainment is concerned, we are trying out a new venture in the shape of a buffet dinner followed by elective courses in theatres, night clubs, etc. We trust this will meet with your approval.

Naturally our thoughts at present are on the terrible conflict raging in the world and its effect upon our lives and national economy. With this thought in mind we have devoted part of our program to our national health program and have been fortunate in obtaining some rare talent. You will hear from these gentlemen presently.

Perhaps in our Medical Departments we feel the greatest repercussions in our constantly changing lists of field Examiners. Those companies who have been concentrating on the younger men, as my own company has done, have found that the induction into service of the reserve corps has kept us busy replacing the inductees. This will continue for some time and the question of competent Examiners in the rural districts is getting serious. All we can do is string along and replace as rapidly as possible our depleted ranks and stretch the good-nature of our remaining Examiners to the limit. So far we have no reason to complain of the co-operation received.

Perhaps the most momentous episodes of the year so far as the Association is concerned have been the retirement of Dr. Arthur Hunter from the Chairmanship of the Joint Medico-Actuarial Committee, and the death on May 17 of Dr. Oscar Rogers, a member of this Association since 1893. The collaboration of these two men in forming sound underwriting practices through their original contributions over a long period of years has furnished a ground work, the value of which can never be over-estimated, and has placed in the hands of their successors a mighty instrument for the future. The work of Rogers and Hunter will always mark the beginning of a new era. To Dr. Hunter: May you enjoy for years to come a life of well deserved happiness! To Dr. Rogers: Peace to his ashes!

It is my hope that further investigations by the Medico-Actuarial Committee may be carefully planned in advance by consultation between the actuaries and medical directors and that some way may be found to make the material more homogeneous, for after all, the results of medical operations are never more precise than the least precise value which enters in to the computation, so that an experimental value cannot be more precise than the least precise data on which it was based. This was plainly shown in our blood pressure investigation of 1929, where data collected haphazard gave some startling results. We must look to the American Heart

Association and our own procedures now advised by our Blood Pressure Committee in taking blood pressures for insurance purposes to furnish us in a few years with new data which may well be most significant.

The general mortality in the United States continues to be good, thanks in a great measure to the sulpha drugs in the acute infections and respiratory diseases. Enthusiasm is still running high over these combinations but signs of a rational curve are appearing. There is much more pioneering to be done. The salutary but unfortunate lesson in the South has taken the drug from proprietary counters. We must not let the enthusiasm of some of our lay underwriters take credit for the low mortality in the United States, give medical advance its due.

With the advances in nutrition through the vitamins, etc., we are already beginning to see bigger and better children and young adults. We may have to change our height and weight tables in this group. In the elders many a case of neuritis, keratitis, paralysis agitans, and other evidences of mal-nutrition are being corrected.

There is nothing startling to report on cancer.

Tuberculosis is keeping down.

Much to our crying shame automobile deaths and maiming is on the upsurge. We weep over the inhuman bombings of European countries but we match that record in our automobile accidents in the United States with a dry eye. How long are we to tolerate this. We have laws enough to stop it. How can we arouse the people. Familiarity apparently breeds contempt. The first suggestion is law enforcement.

With the development of plastics many a new industrial field has been opened and probably some new types of industrial disease. Careful watch must be kept on this new venture.

The recent work done with the electron microscope has already given us some startling results and we can expect much from it in the future. The cyclotron has done advanced work in making new material for use in cancer treat-

ment. Financial aid to worthy institutions for health work and research may well be a part of our insurance program.

All in all, our past year has been one of medical progress. The Americas will have to carry on the brunt of medical advance for some time, even after the war ends. We are sufficiently equipped to do this on this continent, in brains, laboratories, hospitals, and research centers. May the good work go on without interruption!

The American Life Convention has been a sister organization for some time. It is always a pleasure to welcome the Chairman and Delegates from the American Life Convention. This year, we are particularly fortunate in having that genial southern gentleman from Louisville, Kentucky, Dr. W. F. Blackford, to bring us the greetings from the Medical Section of the American Life Convention. May I present Dr. Blackford!

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DR. BLACKFORD—Mr. Chairman, and Members of the Medical Directors Association of America. As a member of this Association I am indeed happy to be here this morning. These meetings are always profitable and enjoyable to me.

As a member of the Medical Section of the American Life Convention, I bring you their best and sincerest greetings for a successful meeting. I have been a member of this Association for some fifteen to twenty years, and I have been a member of the American Life Convention since its infancy. As a member of these associations over this time, I have had the extreme pleasure from year to year of observing the growth and development of admirations and friendships of these associations and their members. The ties and friendships which have developed will never be forgotten and are for the betterment of our respective obligations to our respective companies.

In these days of cold economic, military, and political activity over the world, even nations need unity to exist.

As Chairman of the Medical Section of the American Life Con-

vention, it is my extreme pleasure to extend to this Association, its members, wives, and guests, an invitation to meet with us at the Broadmore Hotel, Colorado Springs, June 4th, 5th and 6th this coming year that we may further extend our friendships and profit for ourselves and our companies by participating in our program at that time. This is a beautiful meeting place at the foot of Pike's Peak, and with our rapid transportation facilities it is in reach of all of us. Our Program Chairman, Dr. Hutchinson, and our Secretary, Dr. B. F. Byrd, assure us of interesting and profitable subjects for discussion and you are cordially invited to join in these discussions as well as to participate in our social functions. Our program of scientific papers and their discussions will be in the morning meetings only; the afternoons will be devoted to recreation, golf, bridge, etc., or intermingling with other members of the Association and their guests. There will be papers presented by outstanding medical authorities and I sincerely trust you will arrange your program so as to be with us at that time. You will be more than welcome.

PRESIDENT CRAGIN—Sherman made one wise remark, that war was hell. War has interrupted part of our program this morning. We were to have Dr. John L. Rice, who has recently been elected President of the American Public Health Association, with us this morning. Unfortunately, Dr. Rice has been called to Washington, and, fortunately for us, we have his assistant to pinch-hit for him, Dr. Edwards.

Dr. Edwards was formerly connected with the National Tuberculosis Association. He has been Director of the Bureau of Tuberculosis of the New York Board of Health since 1934. At the present time he heads the civilian defense program of the New York City Board of Health. He is an outstanding figure in health programs, with particular reference to tuberculosis, and is one of the leading men in this field today.

It is with great pleasure that I introduce to you Dr.

Herbert R. Edwards of New York City, who will speak to us in place of Dr. Rice.

* * * * * *

DR. EDWARDS—Mr. Chairman and Members of the Convention. Dr. Rice regrets very much at not being able to be with you this morning, because, obviously, your work, your aims and objectives are quite similar to ours, and I am sure that he would have been glad to be here, if possible.

He did not leave a manuscript for me to read to you. If I get into too much tuberculosis, I hope you will forgive me. However, I have been associated with Dr. Rice for fourteen years. We have gone through a great many trials and tribulations in the Public Health Administration, and through all of that I think we probably have come out to have a number of ideas that are pretty much alike. His title, that of "Public Health Today", is one that I am sure I have some ideas on, and I shall try to give you the ideas that I think we share in common, and which he probably would have tried to give you this morning.

At the present time, due to the national emergency, we are considering defense. Most of the things we do are now being predicated on defense. It is true that we have to be opportunists now and then to get an opportunity to do some of the things that we have wanted to do for a long time. However, in the field of Public Health, many of the things that we will do at this time are things that have been done before. We only hope that we will be able to do some of them a little bit better, and get a little more co-operation from the public in accepting some of the things that we know is for their good and benefit.

This is the 75th Anniversary of the New York City Department of Health and during these many years there have been many changes and remarkable progress in the prevention of disease and development of programs for the betterment of public health in New York City.

In the early years, such diseases as Small Pox, Cholera,

Yellow Fever, Typhus and Typhoid, were problems of major concern. There were epidemics upon epidemics taking a great toll of life. Today these diseases are practically under control. As we look back over these years we can note many other accomplishments. In the year 1940, the City of New York registered its lowest rates in infant mortality, tuberculosis, diphtheria, pneumonia, typhoid, automobile accidents, whooping cough, measles and other diseases. As satisfying as these accomplishments may be, this is no time to dwell on the past but rather to sharpen our focus on the problems of today as well as those of tomorrow. The leading causes of death in New York City at the present time are heart disease, cancer, accidents, cerebral hemorrhage, tuberculosis, nephritis, pneumonia, diabetes, diseases of early infancy, diseases of the liver and gall bladder, many of which are definitely controllable. Some may be eradicated and others will have to be endured until science has led the way in showing us the cause and, thereby, the method of prevention. Public health today is far different than in the years past. We have now accumulated a great mass of data relative to disease and death with the consequence that this material is now being more carefully analyzed to determine its relative significance insofar as age, sex and race are concerned. Also the modern Department of Health is concerned with an analysis of its procedures. Yet, undoubtedly there is going to be difficulty in securing the funds and financial backing for much of this work in comparison with other defense activities. It is one thing to shift from making automobiles to making tanks, and it is quite another thing to shift some of that money to the channels of Public Health. For many years, we have been like the proverbial sheep, one following the other and it is high time that more consideration be given to the value and productiveness of accepted procedures. This problem deals not only with procedures per se, but with the personnel involved in public health administration. The positions in public health require training and considerable technical skill which is not ordinarily available to all physicians who have received their degree of

medicine. Furthermore, our knowledge is not static and requires constant revision and brushing up to keep all personnel up to the highest standard of attainment. It is time that steps be taken to drop some of those procedures of the past that have proven to be unproductive and perhaps unwarranted. In the case of measles, there is no evidence to indicate that we have effected any real control by methods used at the present time. The Department of Health has, therefore, raised its restrictions on excluding children from school when a case exists in the home. This one step will result in additional allotments from the State of approximately $300,000 a year on the basis of attendance.

In the field of tuberculosis, we have found that the disease occurs in certain periods of life and that it is unnecessary to institute routine supervision at the age period between 3 and 10. This procedure, with the routine x-raying of all persons suspected of tuberculosis, has represented a net saving of 6,000 man hours of labor a year in our services, or $25,000.

In the field of tuberculosis, in which I have been more directly concerned, we have for a number of years been carefully scrutinizing and reviewing the work that we have been doing, and we have come to the conclusion that there are a great many things that we have been doing for years that are entirely unproductive, entirely unnecessary. After all, in the control of tuberculosis there are two important factors. First is to discover the case. Without knowledge of the case, it is impossible to control the case. Therefore, our first emphasis should be on case finding, and that I should say, irrespective of whether or not we have enough hospital beds available to take care of the cases found. Certainly we can never hope to have the beds if we don't demonstrate the need.

We have reviewed our tuberculosis program, and we have found that the routine physical examination, the routine history that is taken and can be taken in the average public clinic, has added nothing whatsoever. We have found that the important thing is to have an x-ray. Therefore, we have provided in our clinics an x-ray unit, and every individual that comes in is

x-rayed. We cut out doing all the routine history work. We have stopped supervising children between the ages of 3 and 10, because, as you know, nothing significant happens during that particular time except in a rare case. Those changes alone have resulted in a net saving of 6,000 man hours of labor a year in our one department, valued at some $25,000. We have not returned any of that money to the city treasury, nor have we dropped any of the staff, but what we have done has been to give each physician in the clinic a real job to do.

I have often thought in the years that I have worked in the tuberculosis field how stupid we were to go through the formalism of thumping a chest which was, after all, only a formalism, yet it was necessary at a time when we felt we should educate the patient, as well as the physician, that to examine a chest one must strip to the waist. I can well recall my experience in Virginia several years ago in which we had a three-ring circus. We had several nurses. They would spend several weeks working up a clinic. I would come in and hold the clinic. They would remain two weeks to follow up. Many days I would go through the shadow-boxing of examining 75, 80, or even 90 people.

You know as well as I do that after the first fifteen perhaps I didn't hear anything, yet we did make every person strip to the waist, and it was for this one purpose only, that of educating that individual that if he ever went to his doctor and had a chest examination, he was to take off his overcoat; it couldn't be done otherwise. I think we have come far beyond that stage in the development of our tuberculosis.

So that in that one step, in the saving of facilities, we have been able to increase the number of individuals who come into our clinics.

From the standpoint of an industrial policyholder, it might be of interest to you to know what we have done in part of our survey work. We have had the opportunity through a survey program here in New York City to cover at the present time something like 400,000 apparently healthy individuals, and the amount of tuberculosis that has been found has ranged any-

where from a tenth of one per cent to as high as five and five-tenths percent among certain selected population groups. That has been very interesting; it has been very productive, but we have seen more and more the need of putting it on a community basis. Therefore, in one of our health center buildings we have made it possible for all persons on relief to come in at a given time, in the forenoons, and to have an x-ray. We have had the co-operation of the Department of Welfare. We have, through their field staff, induced these people to come in voluntarily. It is rather encouraging to note that approximately 65 per cent of the persons above 15 years of age on relief in that district have come in voluntarily for an x-ray, and we have found 2.8 per cent of them with evidence of clinically significant tuberculosis.

If we could apply that same formula to the entire relief population of New York City, which is now at 15 years of age estimated at 300,000, we would probably find over 8,000 new cases of significant tuberculosis that are in need of supervision. We couldn't put them in hospital beds, but we would know them, and if health education has ever had an opportunity, we could do much at that time to do something to probably prevent the spread of that disease to others.

There is also a feeling abroad that we have some definite method of controlling or curing poliomyelitis and influenza, yet there is no method today that can be looked upon with assurance. Thus, it is important for us to reevaluate all of our procedures in every branch of the field of public health to determine primarily whether the procedure set up is accomplishing a purpose, and furthermore, whether it's being done within a reasonable cost. There is a limit to the amount of money that should be spent for the detection or control of a given disease. We have often heard this statement from Public Health Officials, that any amount of expense is necessary if you find it a case, be it tuberculosis, syphilis, or whatever it may be. That may be true if it is my family or your family, but it is not true, in my opinion, when we are dealing

with tax funds and we have a limited budget that must cover a large number of people.

We also feel that Health Education has made tremendous progress during recent years, yet it is clearly obvious that all of our literature is not good, our radio talks do not get across and many of our exhibits are of doubtful value. This field is one of the most important in a public health program, for we must forever strive to secure the co-operation of the public if we are to create a healthy community. It cannot be done by legislation but must be done through education, not only to the masses but to physicians, nurses and other technicians in the field. In all of these matters the Department of Health should take a leading part and increase its activities at the present time. The problems in public health today, as well as those in the future, will show certain variations as time passes. However, there are certain problems that we can select for emphasis that will yield handsome returns for that effort.

At this point I would like to mention a few of the things that we are doing in more or less a directly defense nature. We have felt that an opportunity was afforded through the draft selective service system to really accomplish something in tuberculosis and social hygiene; consequently, when the first call was made, we offered our service staff and facilities to the Surgeon-General of the Second Corps Area to provide a routine x-ray for every man that went through the induction center. At about that time, we had discovered that there had been an order issued from the Adjutant General saying that such services could and should be paid for by the Corps Area Surgeon; consequently, as of January 15 of this year, we were able to get the Corps Area Surgeon in this particular Corps Area to take over the expense of the x-rays and also to assign physicians qualified in roentgenology and chest diseases to the staff to read the films. At the same time the Department of Health continued to co-operate with the Army officials by having all of those persons who had suspicious or

significant lesions, who were automatically rejected at the induction center, referred to our central chest clinic where we at once started a complete work-up and study of that case.

It is rather interesting to note that about ten percent of those who were thus cleared were certified back to their draft board and to the induction center as suitable for military training. Furthermore, we not only followed through with that particular case to see that he got under his doctor's care, got into a hospital if necessary, but that if he had no physician, he could go to a clinic.

We didn't stop just with the individual. We naturally went on to the contacts in that home, we made a complete job of it, so that in the past seven or eight months, or close to a year now, well over 100,000 persons have gone through this examination. The army has taken the major part of it, but the Department of Health stands ready to follow through to take these early minimal lesions; they may not be early, but at least minimal, to supervise them when there is time to accomplish something.

In a similar way we offered the services of our laboratories to the draft boards so that every man that was registered had an opportunity to have a Wassermann test. That placed a terrific load on our facilities, but we have been able to adjust it and to carry on that important work. There again we not only do not stop with making a diagnosis, but we follow through on that individual. I am sure you can appreciate what that must mean in the long pull.

We also are working now on a nutrition program. We believe that nutrition is one of the basic things. We do not think it is booked to hang everything on, nor is it an end in itself. It is a thing that must go along with a balanced Public Health program the same as we have a balanced diet. It has a part to play. We hope to see developed here in New York City a nutrition program that will be citywide in extent, not one that is sponsored only by the Department of Health, or any city department, but a City of New York program

which will include every organization of any size or nature or character in the city.

We hope that they will all come in with us and carry through on this thing. We believe that we can do a great deal to improve life and living and prolong life.

There are other things that I think we should consider in our Public Health program today and which we are giving consideration to, to step up a little, if possible, to make more important. We have accomplished a great deal in New York City in the immunization for diphtheria, but as occurs in so many instances, the thing that we have is a thing we least appreciate, at least we don't realize what its struggle has been to attain that. Therefore, there has been a tendency for mothers and parents to be a little bit negligent about having their children immunized. That is a very important thing. In these times of stress when we don't know whether we will be invaded, whether there will be mass evacuation, or there will be bombing, or various other things, it is vitally important that every infant, every child, be sure that he is protected against diphtheria. That is something that can be done. We are emphasizing that part of our program, to have every infant immunized. We are not a hundred percent and we should be a hundred percent. The same is true of smallpox.

Now, to step these things up, our laboratories are provided, for example, with enough antitoxin or enough vaccination for smallpox for at least a million persons. We could easily step that up. All of our biologicals, all of our materials are being stepped up and we have a stock on hand so that if the occasion arises, we will be in a position to give this protection.

The death rate from tuberculosis has been reduced by approximately 75% in the past thirty years but our progress in further reduction is too slow when we consider the knowledge at hand today. The x-ray is now considered as of far better diagnostic value than the physical examination. The x-ray may be had at reasonable cost and there is no legitimate reason why our efforts in case finding should not be increased ten

fold or more, because it is only when the case is discovered that we can hope to control the further spread of the disease.

Great progress has been made in the past few years in the control of syphilis and gonorrhea. Here, again, we have techniques and facilities to diagnose and treat cases. There is need for a more extensive program to reach all walks of life—in the home, schools, industry, etc.—to discover cases and apply the indicated treatment.

Diabetes is a disease that is definitely controllable, yet we are not utilizing all of the facilities possible in its detection and treatment. Accidents now hold third place as a leading cause of death, the majority of which could be prevented, and its loss to industry alone very materially affects its productive outlook. Accidents, due to street traffic, are tending to increase, and likely with the conditions we are facing today and for the next year or so, will probably show an increase rather than a decrease. There must be more collaboration in the working together of Health Department, Police, Red Cross, accident prevention committees and all others who are interested to aid in this problem.

Industrial hygiene is a field practically untouched. There is need for definite medical services in every industry and particularly at this time when the national defense depends so definitely upon the health and well-being of all the people. It would seem to me if there was ever a time when we had an opportunity to cash in on industrial hygiene it should be at this time. Some efforts have been made with labor organizations. We in our surveys, for example, in tuberculosis have been able to reach a large number, some twenty-five to thirty thousand people, through the unions, and we have been trying to get them to work more with the employer. They, of course, are skeptical of the employer if he says he wants an x-ray of a man. "Suppose he does and I lose my job?" That job is always in the balance. But there is some light on the horizon. One case will indicate my reaction on that.

In a union we surveyed recently, we found a case that was

in need of hospitalization. That woman had joined the union only a few months before. She had just come under the deadline where she was entitled to all the benefits and emoluments of the union. She was a rather early case. The case was sent to the hospital. The leader of the union came around and said, "Listen here, if I had had all of our new applicants for our union x-rayed for the last two years, it wouldn't have cost me as much as it is going to cost me to take care of this one person," and he immediately went over to the union health center and arranged for all new applicants in that union to have a routine x-ray examination.

I don't think the problem is as difficult as we are wont to believe some times. I think now is the time that we should redouble our efforts to try to get the co-operation of both labor and industry to carry this thing forward.

The problem of nutrition is now taking on immense proportions. We are no longer concerned merely with the definitely mal-nourished individual as indicated by clinical signs, but rather with a large mass of the population who have latent nutritional defects that should be corrected. If, as Surgeon General Parran has recently stated, this country may be faced with the possibility of rationing of food, this problem will become even more acute. It is vitally necessary, therefore, that we take every step possible to see that all the population has enough of the essentials for a balanced diet. It is not enough to get more money for the individual. We have got to begin to teach the individual how to use that money and what types of food are best for him. One of the greatest experiments in this field was recently reported in the press by Col. R. A. Osmun of the Army Quartermaster Corps, in which he stated that the men in the service today are getting a well balanced diet and he commented that many who lived at home on lean meat, potatoes, and coffee or on hominy grits, fat pork, molasses and little bread are now learning what life is like with a balanced diet. As you noticed in The New York Times, there was a write-up on

the editorial page this morning pointing out that these individuals who had never had a balanced diet are now learning to like vegetables and salads and are eating the things really desirable.

Housing is also a matter concerned with the public health. Great strides have been made but there is a clear indication that further effort should be made to provide sanitary, safe and clean housing for the greater masses of our population.

The field of mental hygiene has hardly been scratched at the present time. This also is a matter concerning the public health and one in which the Department of Health should take a more active part in the future.

During the past years, we have spent millions of dollars in dental work, yet it is reasonable to assume that until we have research in the underlying causes of dental decay, we will not be able to put this program on a practical basis as is indicated.

Diseases of old age present one of our greatest problems. It is estimated that by 1980 our population at 65 years and over will double that of today. We know very little about the diseases that occur in that period of life. It is encouraging, however, to note that the National Institute of Health, as well as other agencies, are now devoting an increasing amount of effort to solving these problems. We have in New York City, as you probably know, a hospital of some 1,200 beds devoted to this very problem.

On the question of research, it is of interest to report that the Board of Estimate and the City Council has authorized the City to enter into a contract with the Public Health Research Institute of New York City, Inc.—a corporate structure which is to be a scientific non-profit organization entirely devoted to obtaining for the City the best available biological products and advanced skills and procedures for combating disease and epidemics which occur or may occur in New York City.

In closing, I would like to quote a statement made by Com-

missioner Rice in his Presidential Address at Atlantic City recently as I believe it indicates more clearly than I can state the attitude of the Department of Health at this time. "Health Departments must assume leadership and responsibility for the broader field of public health. Our job should be something more than preventing diseases and reducing death rates. Good health, not mere survival, is of the greatest importance to mankind. Happy and wholesome living should be our goal. The success of our activities for the attainment of a maximum of health for the people will depend upon wise planning, efficient operation and constant employment of practical measures."

PRESIDENT CRAGIN—Thank you very much Dr. Edwards. We appreciate your coming, and as a pinch-hitter, I would put you on my baseball league any time.

The next paper is one that is of interest to us all, and comes from the joint efforts of Dr. Robert M. Daley, Dr. Harry E. Ungerleider and Dr. Richard S. Gubner of the Equitable Life Assurance Society. The title of their paper is "Prognosis and Insurability of Hypertension With Particular Reference to the Electrocardiogram." The paper will now be read by Dr. Ungerleider.

"PROGNOSIS AND INSURABILITY OF HYPERTENSION WITH PARTICULAR REFERENCE TO THE ELECTROCARDIOGRAM"

ROBERT M. DALEY, M. D., MEDICAL DIRECTOR
HARRY E. UNGERLEIDER, M. D., ASSISTANT MEDICAL DIRECTOR
RICHARD S. GUBNER, M. D.
The Equitable Life Assurance Society

The many papers on hypertension which have been presented before the Association would, at first, appear to call for an apology for this presentation, but on the other hand, the numerous studies in themselves indicate the importance and manifold unsettled aspects of the problems of hypertension. Despite tremendous strides in medicine, the expectation of life today in the middle and older age groups, at which ages hypertension is so prevalent and a leading cause of death, is practically identical with life expectation at the beginning of the century[1]. With the shift in population toward increasing years and improvement in mortality from infectious diseases, such as pneumonia, the problem of hypertension is bound to assume even greater significance.

Broadly, hypertension is important for two reasons; as an indication of an underlying disease, and because of the deleterious effects of the high blood pressure itself. Elevation of the blood pressure can result from a great variety of causes. These have been classified into five major groups; 1) renal, 2) endocrine, 3) vascular lesions, 4) central nervous system disease and 5) essential hypertension of unknown origin[2]. In the past few years, there has been an attempt to identify essential hypertension with experimental hypertension produced by renal ischemia[3]. While the impetus from this line of investigation has resulted in the finding of primary renal disease in a certain number of cases which would otherwise have been regarded as examples of essential hypertension, the vast majority of cases of essential hypertension, however,

show no evidence of any renal disease whatsoever, even when studied by refined functional tests[4]. The renal blood flow is frequently reduced in hypertension[5], but there is no proof that this is the cause rather than the effect of the circulatory changes. On pathological, experimental and clinical grounds, it seems quite dubious, as yet, that renal ischemia is the underlying mechanism of "essential" hypertension.

The cases of hypertension where the etiology is known and can be ascribed to renal, endocrine or other organic disease constitute but a very small fraction of the total number of cases with elevated blood pressure and are of little practical moment in insurance selection, for the high mortality of the underlying disease precludes any insurability. The problem with which we are concerned is that of essential hypertension which constitutes the vast majority of cases with elevated blood pressure. While the cause of essential hypertension remains obscure, much is known of various clinical aspects of hypertension. The purpose of this study is to apply our knowledge of the prognosis of hypertension to the problems of insurability.

Medico-actuarial insurance studies have contributed much to various phases of hypertension, but it has impressed us forcibly that insurance medicine and the actuaries have considered hypertension not as a disease or symptom of a disease with a natural and largely predictable course, but arbitrarily as merely a question of numerical units of blood pressure. Hypertension is injurious, not only because of the direct consequences of elevated arterial tension, but because over a period of years cardiac enlargement and heart failure ensue, and degenerative arteriosclerotic changes occur in the coronary and other vital vessels. The mortality from heart failure and degenerative vascular changes which result from long-standing duration of hypertension, *without regard to its degree*[6], is far greater than the mortality from such causes as malignant nephrosclerosis and cerebral hemorrhage, which can be attributed more directly to marked elevation of the blood pressure, although even here degenerative changes in the arterioles

caused by hypertension existing over some period of time are an important factor. Sudden extreme rises in the level of blood pressure leading to rapidly fatal malignant nephrosclerosis or apoplexy may occur at any time in the course of hypertension, of whatever etiology[7], but ordinarily essential hypertension has a characteristic evolution over a period which may vary from 10 to 15 years or more, with the ultimate development of heart failure, coronary thrombosis or other vascular complications and, in a smaller number of cases, uremia. It is evident, therefore, that a most important consideration from the viewpoint of insurability is the proper evaluation of the duration of the hypertension, i. e., the stage of the disease.

The stage of hypertensive disease may be revealed from several approaches. The criterion which has been employed in life insurance medicine almost exclusively is the blood pressure reading. The increases in mortality with each increment of blood pressure which have shown up consistently in mortality studies have tended to lull us into a false sense of finality and confidence which is, however, abruptly disspelled when the many vagaries of blood pressure are considered. The progressive increase in mortality with rising blood pressure is attributable to the fact that in a very general way, the blood pressure tends to become more elevated with increasing duration of the hypertension. This is by no means the rule however and to quote an authority[8], "In some instances the blood pressure rises rapidly or more often slowly, but progressively as the patient is watched for years. On the other hand, in very many patients the height of the pressure does not change notably from that found at the first examination, even though it is followed for years".

A further objection to evaluating hypertensive subjects solely by blood pressure readings is the extreme variability of the blood pressure. To quote again[9], "The instability of the blood pressure in essential hypertension will often lead to normal readings of the blood pressure if the patient sits quiet for from 5 to 20 minutes. This occurs even in patients with widespread

vascular changes. 56% of 76 unselected, untreated cases exhibited this finding". The diastolic pressure fluctuates proportionately as widely as the systolic pressure[10]. Even among subjects with hypertension who are accustomed to frequent blood pressure determinations and trained to make their own readings, it is found that readings are far lower when made at the patients' home, than in a clinic in the presence of a physician[11]. We must recognize that there is an element of sophistry in the mechanical averaging of a number of readings in various fashions, and so the importance of determining the extent of organic changes which have resulted from the hypertension becomes apparent. The extreme fluctuations in blood pressure also work unfairly both to the applicant and to the company. Subjects who are aware that their blood pressure is elevated are likely to arrange examinations under circumstances most favorable to themselves and to prepare themselves by rest and other less natural means. On the other hand, applicants may be examined under conditions of stress and readings may be obtained higher than their usual levels. Fluctuations in blood pressure cause particular concern in the borderline normal group. *It is becoming increasingly recognized that subjects whose blood pressure rises to abnormal levels under any circumstances are predisposed to subsequent hypertension*[12-14] and several tests employing various pressor stimuli have been devised to measure the reactivity of the blood pressure as an index of potential hypertension[15-25]. These tests are not likely to find application in insurance medicine, because while some of the procedures are simple, such as breath holding or immersion of the hand in cold water, meticulous attention to details is required[26] and a basal level must first be attained. In effect, the initial blood pressure reading on physical examination may be regarded as a pressor test and has been recognized as having the same significance[14,27]. It might be well to recommend as a general rule that the blood pressure be taken at the start of an examination and if elevated should be repeated toward the end of an examination when the applicant is less likely to be apprehensive. It would also be advisable to emphasize that all

readings must be reported, and that the applicants' demeanor and circumstances attending the examination should be noted.

Apart from the difficulties occasioned by the variability of the blood pressure, various other factors cause difficulty. For example, changes in posture may have a marked influence on the level of the blood pressure. Occasionally, the blood pressure may differ not inconsiderably in both arms, even in the absence of vascular or other pathology. Even the accuracy of blood pressure readings, particularly of the diastolic level, is frequently to be questioned as was strikingly brought out in a study on the factors of error in blood pressure readings[28]. Finally, an abnormal blood pressure may have widely divergent significance. For example, a blood pressure of 170/80 may be due to such varying causes as advanced arteriosclerotic changes in a subject with long-standing hypertension, an emotional reaction in a normal individual, heart block, Grave's disease or aortic insufficiency.

These factors, among others, indicate the inadequacy of depending solely on the blood pressure reading in evaluating insurability in subjects with hypertension.

Examination of the urine is of little value as an adjuvant in interpreting high blood pressure, except to distinguish those cases of hypertension due to glomerulonephritis or other renal disease. Albuminuria occurs in a comparatively small proportion of cases of hypertension and while marked albuminuria is a poor prognostic omen, there in no correlation between albuminuria and the course of hypertensive disease. Impairment in renal function does not occur until very late in the course of the disease and only a small fraction of cases with essential hypertension progress to renal insufficiency.

Changes in the retinal vessels occur regularly in essential hypertension and have been used to grade hypertension into 4 stages[29]. "The presence of retinal arterioscleroses or arteriosclerotic retinopathy indicates that the hypertension has been present for a considerable period[30]." However, the majority of cases of hypertension are seen in a stage when changes in the retinal vessels are minimal[29]. While in the

hands of the expert ophthalmoscopist, examination of the retinal vessels is of great value, interpretation of the retinal changes is subjective and cannot serve as a procedure to be generally employed as a strict criterion of insurability.

Two changes occur regularly, however, with increasing duration of hypertension which can be evaluated objectively, and which we feel are serviceable as prognostic guides in assessing insurability; 1) left ventricular hypertrophy and 2) eventually arteriosclerotic changes, particularly in the coronary vessels and the aorta, are invariable accompaniments of elevated blood pressure of long standing. Hypertrophy antecedes the arteriosclerotic changes somewhat, as is indicated by the finding of cardiac hypertrophy, but relative freedom from arteriosclerosis, in cases of hypertension due to renal disease[6]. This may be attributed to the relatively brief duration of the hypertension which occurs late in the course of glomerulonephritis. It is of importance to note that arteriosclerotic changes are more closely related to the duration than to the degree of hypertension, and in a recent study it was found that "the incidence of marked coronary disease is the same in cases of mild and severe hypertension[6]".

Hypertrophy and arteriosclerosis of the aorta and coronary vessels are best revealed by means of the X-ray and electrocardiograph. The present study is concerned with an investigation of the significance of hypertrophy and arteriosclerosis in subjects with hypertension, based on the hypothesis that since these changes are indications of hypertension of long standing, they may be used advantageously to segregate subjects with hypertension into different stages of the disease.

Hypertrophy of the left ventricle can be determined by means of the electrocardiograph and X-ray, which also serve to reveal arteriosclerotic changes in the coronary arteries and the aorta. Arteriosclerosis of the aorta is of greater significance than arteriosclerosis of the peripheral arteries, since it is more closely associated with coronary artery disease. The roentgen findings

in arteriosclerosis of the aorta are quite characteristic and consist of elongation, tortuosity, increased density and calcification of the transverse and descending arch. The ascending aorta is not so frequently involved by arteriosclerosis, although it may become somewhat dilated, perhaps to act as a larger reservoir in consequence of the arteriosclerosis of the remainder of the aorta. The systolic murmur over the aortic area, which is frequently present in subjects with hypertension as arteriosclerosis, is not necessarily due to sclerotic changes in the aortic valve or aorta, but is frequently due to dilatation of the ascending arch which often accompanies arteriosclerosis of the aorta. Numerous methods have been proposed to detect dilatation of the aorta, none of which is too satisfactory.

The criterion employed in the present study to evaluate arteriosclerosis of aorta is the measurement proposed by Dr. Sheridan[31], i. e., the transverse diameter of the vascular pedicle related to standards of height and weight, as employed in predicting the transverse diameter of the heart[40]. This does not distinguish dilatation from arteriosclerosis, since increase in the diameter of the vascular pedicle can result from dilatation or tortuosity of the aortic arch, but it does indicate whether the aorta is normal or abnormal. A study of 372 teleoroentgenograms of normal subjects indicates that the prediction standards proposed by Dr. Sheridan apply equally well to the teleorcentgenogram and we have used his standards based on weight, height and age with an allowance of 10% above the prediction value*. The influence of age was found to be relatively unimportant as a determinant of the transverse diameter of the heart, but it is a most important consideration in the transverse diameter of the aortic arch, since there is a progressive increase in this measurement beyond the fourth decade.

* 82.3% of normal subjects fell within 10% above the predicted value. Diameters in excess of 15% above the predicted value may be considered as almost certainly abnormal (provided the X-ray is made with the subject properly centered), since 92% of normal subjects fall within this range. Analysis of the 372 normal subjects showed that the influence of height was relatively negligible, the correction from a range of 59 to 76 inches being less than 1 mm. It was found that a formula based on age and weight yielded practically identical results with the standards prepared by Dr. Sheridan which consider height as well as age and weight. This formula is Aortic T. D. equals .30 age plus .14 weight plus 23.86.

For the evaluation of heart size or, more specifically, left ventricular hypertrophy, three X-ray measurements were employed. These included the relation of the apex to the left midclavicular line, the transverse diameter of the heart as related to height-weight prediction standards, and the cardio-thoracic ratio.

Electrocardiographic changes due to coronary disease are well known, but less attention has been paid to use of the electrocardiograph than the X-ray in detecting hypertrophy. In a general way, the pattern of left ventricular strain is recognized consisting of left axis deviation in association with high voltage of the Q. R. S. complex, depression of the ST segment in lead 1 (and 2) and T wave abnormalities in leads (1 and 2). While the association of a characteristic pattern of electrocardiographic changes with hypertrophy of the left ventricle has been noted by many investigators[32-36], beginning with Einthoven's early observations, specific criteria, however, have not been established. We, therefore, undertook a study of a large group of subjects who had been examined in the Diagnostic Laboratory of the Equitable Home Office with the object of determining what electrocardiographic changes, in conjunction with left axis deviation, could be considered indicative of left ventricular hypertrophy.

The material for study included three groups of cases:—

A. 460 cases with left axis deviation in whom the blood pressure readings were always below 140 systolic and 90 diastolic, and in whom there was no record nor finding of a cardiac impairment.

B. 380 cases of applicants with hypertension with left axis deviation in whom the blood pressure in all readings was above 140 systolic and 90 diastolic.

C. 100 disability cases of advanced hypertension with left axis deviation.

Group "A" was taken as "normal" and Group "C" as "hypertrophy". Group "B" consisting of applicants with hypertension may be regarded as a mixed group in which a

certain proportion had hypertrophy, while the remainder were normal. A study was made of voltage and of changes in the ST segment and T waves. In the consideration of voltage, two questions were investigated:—

1. What combinations of measurements of the R and S waves in leads 1 and 3 is the most efficient criterion for the separation of those with normal hearts from those with hypertrophied left ventricles?
2. With this criterion determined, what is the dividing line between normals and abnormals. Five combinations of the R and S waves in the standard limb leads were investigated:—
    1. Amplitude of R1
    2. Amplitude of R1 plus S3
    3. Higher of R1 or S3
    4. Higher of (R1 plus S1) or (R3 plus S3)
    5. (R1 plus S3) minus (S1 plus R3)

The differences are slight, but criterion "2" appears most efficient, followed by "5" and then by "3" or "4" indifferently. Criterion "1" seems poorest. Hypertrophy of the left ventricle may, therefore, be considered to be present in subjects with hypertension when, in conjunction with left axis deviation, the sum of the amplitude of the R wave in lead 1 and the S wave in lead 3 exceeds 25 mm. 99% of normals with left axis deviation are below this value; whereas, this is exceeded by 51.9% of cases with hypertrophy of the left ventricle.

The Group B cases can be considered a mixed group consisting in part of those in the early stages of essential hypertension and in part of those of the later stages of the disease. The relative proportions of these two classes, of course, controls the confidence with which it can be asserted that a case exhibiting a given amplitude of the R1 + S3 criterion has an hypertrophied left ventricle. It may be of interest that 44.2%, 29.2%, and 15.9% of this "mixed" distribution fell above the 95%, 99%, and 99.9% levels, respectively, of the "normals" curve. This shows, first, that the proportion of those in the

TABLE 1

Criterion	95%		99%		99.9%	
	of "normals" are below this point	which is exceeded by this % of hypertrophies	of "normals" are below this point	which is exceeded by this % of hypertrophies	of "normals" are below this point	which is exceeded by this % of hypertrophies
1. R1	13.9 mm.	60.1%	15.8 mm.	42.6%	18.1 mm.	23.5%
2. R1 + S3	22.1 "	67.0%	√25.4 "	51.9%	29.1 "	34.7%
3. Higher of R1 or S3	14.0 "	63.6%	16.1 "	45.7%	18.4 "	27.1%
4. Higher of (R1 + S1) or (R1 + S3)	15.6 "	62.6%	17.7 "	46.0%	20.2 "	27.4%
5. (R1 + S3) minus (S1 + R3)	19.2 "	65.1%	22.5 "	50.0%	26.1 "	33.6%

advanced stages is sufficiently great in this "mixed" group that we may use the levels we have determined with some degree of confidence. Second, it shows that the number of cases with hypertrophy in a group notable only for elevated blood pressure readings is large enough to warrant the trouble and expense of further investigation.

Changes in the ST segment and T waves are frequently observed in hypertension, but are less specific than increase in the amplitude of the Q. R. S. complex, since such changes occur in a great variety of conditions other than hypertrophy. It is generally stated that depression of the ST segment is significant only if it is 1 mm. or more (0.1 millivolt) below the isoelectric baseline. However, review of over 400 normal cases with left axis deviation disclosed none with any noticeable depression of the ST segment in lead 1; whereas, this is very frequently seen in cases with left ventricular hypertrophy. We, therefore, regard depression of the ST segment in lead 1 of as little as 0.5 mm. as suggestive of left ventricular hypertrophy in the presence of hypertension. Flattening of the T wave to less than 1 mm. amplitude likewise is to be considered suggestive of left ventricular hypertrophy when associated with left axis deviation in subjects with hypertension. The depression of the ST segment and T wave negativity in lead 1 is accompanied by a reciprocal elevation of these deflections above the baseline in lead 3.

The changes in the ST segment and T wave described are characteristic, but are less specific of left ventricular enlargement than increased voltage. Similar changes occur in any condition causing left ventricular strain, where the work of the left ventricle is increased without commensurate increase in coronary flow, or wherever a relative myocardial ischemia exists, as in shock, anemia, tachycardia, aortic stenosis or insufficiency, etc. It is likely that the changes in the terminal deflection of the ventricular complex are an expression of a state of chronic coronary insufficiency rather than due to hypertrophy itself, since several factors operate to produce a

relative disproportion between the oxygen requirement of the myocardium, particularly in the deeper layers of the left ventricle, and the coronary flow in the presence of hypertension.

It should be emphasized that left axis deviation alone is of no diagnostic value in hypertension, although it was present in 78% of the cases with left ventricular hypertrophy. Left axis deviation occurs in a very large proportion of normal subjects, particularly those of heavy-set build with transversely placed hearts. Indeed, one reason for the occurrence of left axis deviation in a preponderance of cases with hypertension is that the greater number of these subjects are of sthenic build in whom left axis deviation occurs normally.

Analysis of electrocardiograms in a group of over 350 cases with hypertension showed a close correlation of left axis deviation with body build. Those cases with stocky build almost invariably had left axis deviation; whereas, in those of normal stature, left axis deviation was not observed so frequently. In fact, right axis deviation was present in some cases of asthenic habitus. These findings indicate that axis deviation depends largely on body build and, therefore, left axis deviation alone cannot be considered indicative of "left ventricular preponderance", a term which has inaptly been used synonymously with left axis deviation. Marked left axis deviation, where the Q. R. S. complex is inverted in lead 2 as well as in lead 3, does not occur normally, but is due to myocardial involvement rather than to ventricular enlargement. This has been termed pathological left axis deviation.

Having established electrocardiographic criteria of hypertrophy of the left ventricle, analysis was made of 100 cases with advanced hypertensive heart disease and the electrocardiographic and X-ray findings were compared. Three criteria were employed in estimating heart size in the roentgenogram; the relation of the apex to the mid-clavicular line as seen in the X-ray, the cardiothoracic ratio and the height-weight prediction table for transverse diameter of the heart. In addition,

the width of the vascular pedicle was measured as an estimate of the size of the aorta according to the method proposed by Dr. Sheridan.

The findings are tabulated:— (Table 2)

Electrocardiographic & X-ray Findings in 100 Cases of Advanced Hypertensive Heart Disease*

A

Electrocardiogram abnormal in	69%	X-ray abnormal	78%
pattern of hypertrophy	58%	apex outside midclavicular line	52%
myocardial disease	11%	cardiothoracic ratio above 50%	36%
		transverse diameter more than 10% above predicted	41%
		(aortic diameter more than 10% above predicted	54%

B

Electrocardiogram positive	–	X-ray positive	57%
Electrocardiogram positive	–	X-ray negative	12%
Electrocardiogram negative	–	X-ray positive	21%
Electrocardiogram negative	–	X-ray negative	10%

Several findings worthy of note are: 1) that in advanced hypertensive heart disease, X-ray and electrocardiographic study will reveal hypertrophy or arteriosclerotic changes in 90% of cases; 2) that the electrocardiogram compares very favorably with X-ray methods for detecting left ventricular hypertrophy; 3) that the relation of the apex to the midclavicular line, as seen in the X-ray, is a simple and useful index of left ventricular enlargement; 4) that the cardiothoracic ratio, as has been shown repeatedly[37], is the poorest of the measurements and 5) that the aorta is abnormal in a majority of cases of well established hypertensive heart disease.

It was of interest to find that the electrocardiogram was most often normal when there was no deviation of the electrical axis. Thus, while left axis deviation was present in 78% of cases and no axis deviation in 22%, 15 of the 22 cases with no axis deviation presented normal electrocardiograms. The body build in this group was less sthenic than in the group

* The electrocardiographic criteria were much stricter than the X-ray criteria for enlargement. In only 1% of normal subjects does the amplitude of the QRS complex expressed as the sum of the R wave in lead 1 and S wave in lead 3 exceed 2.5 millivolts, which was the criterion employed. On the other hand, the criteria for transverse diameter of the heart and width of the aortic arch silhouette (over 10% above the predicted value) are exceeded by 15% and 17% of normal cases respectively. Had equivalent criteria been employed, the electrocardiogram would have been abnormal in a relatively much greater percentage of cases.

of 78 cases with left axis deviation. The conclusion to be drawn is that the pattern of left ventricular hypertrophy evolves most readily in subjects with transversely placed hearts who are predisposed to left axis deviation. The electrocardiogram is of less value in detecting hypertrophy in subjects of slender stature. Characteristic changes in the precordial leads have been described in left ventricular hypertrophy, but these were not studied in the present investigation because precordial leads were not made in all the electrocardiograms which formed the material for this report[38].

Returning to the original thesis, a mortality study was carried out to determine whether electrocardiographic criteria for the detection of left ventricular hypertrophy and arteriosclerotic changes would allow classification of subjects with high blood pressure into distinct groups, regardless of blood pressure levels and provide an index of the duration and stage of the hypertensive disease. This study was carried out with reference to the electrocardiographic findings and without consideration of the teleoroentgenogram, although this would have contributed much to the investigation.

The material for the study comprised 424 cases of insurance applicants who were examined in the Diagnostic Laboratory at the Equitable Home Office between the years 1929 to 1941. In practically all the cases, the primary impairment which led to examination in the Diagnostic Laboratory was hypertension, but not of sufficient degree to cause declination. Higher readings which, in themselves, would have dictated declination without further study were found, however, in a number of cases at the time of Home Office examination.

The electrocardiograms of these 424 subjects with hypertension were divided into 4 groups:—

1. Normal electrocardiogram
2. Borderline
3. Pattern of left ventricular hypertrophy
4. Evidence of myocardial disease with or without left ventricular hypertrophy

The over-all mortality results were as follows:—*

Group	Cases in Mortality Study	Actual Deaths	Ratio of Actual & Expected
1.	185	23	186%
2.	97	19	269%
3.	101	50	344%
4.	41	15	375%
All	424	88	271%

The data were tested for homogeneity as to age, build, and blood pressure. They were found to be homogeneous as regards age and build and reasonably homogeneous as regards blood pressure. The average blood pressure readings had a slight tendency to increase from group to group, as shown in the following table, but with the rise among the first three groups being small.

Group	Average Systolic	Average Diastolic
1.	156 mm.	97 mm.
2.	159	97
3.	159	98
4.	163	100

In order to test the importance of the increase in blood pressure, the data within each group and in the study as a whole were subdivided into the five systolic pressure groups 140–149, 150–159, 160–169, 170–179 and 180 and over. The expected deaths within each of these subdivisions for the four groups which have been described above were multiplied by the actual to expected mortality ratio of the corresponding systolic pressure subdivision for the study as a whole. With this crude adjustment, the ratio of increase from the lowest to the highest mortality ratio among the four groups was reduced by approximately one-quarter, but the general pattern of mortality remained relatively undisturbed, with groups three and four being definitely higher than groups one and two. Thus, if the mortality ratio in the group with the normal elec-

* The entrants were followed up in the cases where the policy lapsed and where it was declined or not taken out. The exposure was from the date of the electrocardiogram to the anniversary of that date preceding September 1, 1941. Expected deaths were calculated by a table based on standard medically examined issues for a similar period.

trocardiograms (Group 1) is expressed as 100, the mortality ratios in the other group shape up as follows:

Group	Index of Mortality Ratios	
	Without B. P. Adjustment	With Systolic B. P. Adjustment
1.	100	100
2.	145	126
3.	185	178
4.	202	170

In view of the relative homogeneity of the factors of age, build and blood pressure, the increase in mortality must be considered as correlated with the condition which caused the electrocardiographic changes which formed the criteria for the sampling. It is evident that in subjects with hypertension who present the pattern of left ventricular hypertrophy, as defined by the criteria established in this study, the mortality becomes prohibitive as regards insurability. Even the group with normal electrocardiograms has a mortality of 186%, but this is within insurable limits. The conclusion appears warranted that the evaluation of the duration of hypertension based on the electrocardiogram is a valuable and an important consideration in the prognosis and insurability of subjects with hypertension.

The findings abundantly justify the employment of more detailed study in the presence of hypertension. Since the electrocardiogram was normal in only 44% of 424 applicants with hypertension, it would appear worthwhile to take electrocardiograms routinely whenever the blood pressure is over 140 systolic and 90 diastolic. With the widespread use of portable electrocardiography today, it is quite feasible to have electrocardiograms made in field examinations by competent examiners, when Home Office study is not possible. The applicability of the X-ray is somewhat more limited, but it would appear desirable to take X-rays whenever feasible.

The findings of the present study are not intended to serve as a final basis for action in underwriting, but rather to indicate the need and usefulness of considering the stage of hypertensive disease as judged by evidence of hypertrophy,

and arteriosclerotic changes in the aorta and coronary vessels. Except for the paper of Dr. McKenzie and Mr. Shepherd presented before the 1937 meeting of the Association and the paper by Dr. Bolt in 1934, there has been little or no attention paid to the duration of hypertension. Both these contributions indicated, as does the present study, that the mortality is higher where there is an antecedent record of elevated blood pressure.

Application of this aspect to actual underwriting can be achieved by a collaborative study, such as the 1939 Blood Pressure Study. The mortality experience would be determined for systolic and diastolic levels at various ages as in the 1939 study, but in addition the cases would be separated into 5 groups in each tabulation, i. e., a) cases with no hypertrophy or arteriosclerosis, b) cases with borderline changes, c) cases with hypertrophy, d) cases with arteriosclerosis (coronary arteries or aorta) and e) cases with hypertrophy and arteriosclerosis.

The recommendation for the general use of the electrocardiogram, and the X-ray when possible, may arouse criticism as a refinement which will too stringently limit selection. On the contrary, we predict that in cases without evidence of hypertrophy and arteriosclerosis far more latitude will be exercised in accepting, as insurable, subjects with elevated blood pressure and the field for selection will be enhanced rather than decreased.

The problems discussed in this report constitute but one phase of the factors determining prognosis in hypertension. The effect of age has been investigated, but there are other important constitutional factors which influence the course of hypertension. Thus, hypertension while more prevalent in females appears more benign than in males and it has been reported in clinical studies that the mortality in hypertensive subjects is twice as great in males as in females[39]. Hypertension is more prevalent among urban negroes than in the white population and the disease seems to run a more rapid course, terminating in malignant hypertension or heart failure

more frequently than coronary disease, which requires a longer period to develop. Heredity is another important consideration. The association of diabetes with hypertension markedly accelerates the rate of development of arteriosclerosis and so glycosuria in the presence of hypertension is to be regarded more unfavorably than either of these impairments alone. These questions, among others, all offer fertile fields for investigation. It may be hoped that eventually all these factors may be statistically evaluated and so provide a more scientific and equitable basis for determining insurability in hypertension.

SUMMARY

This study was undertaken with the thought that the employment of blood pressure alone in determining insurability in subjects with hypertension has many short-comings. Essential hypertension has a characteristic evolution over a period of many years and it was felt that recognition of the stage of the disease, i. e., the duration of the hypertension, would be a valuable aid in evaluating prognosis and insurability. The blood pressure is of limited value for this purpose, and various other methods for determining the stage of hypertension are discussed.

Two constant concomitants of hypertension of long standing are left ventricular hypertrophy and arteriosclerotic changes in the aorta and coronary arteries. These are revealed in the electrocardiogram and in the X-ray. Electrocardiographic criteria of left ventricular hypertrophy have not been well established and, therefore, a study was made of 460 electrocardiograms of normal subjects with left axis deviation and 480 electrocardiograms made on subjects with hypertension, in order to establish what specific changes could be considered indicative of left ventricular hypertrophy. Left axis deviation was present in 78% of cases with advanced hypertensive heart disease, but this finding alone is of no value since left axis deviation occurs in a large percentage of normal subjects. However, it was found that left axis deviation associated with increase in the amplitude of the Q. R. S. complex and/or

changes in the terminal deflections of the ventricular complex provide a means for distinguishing hypertrophy of the left ventricle. The amplitude of the Q. R. S. complex was analyzed in various ways and it was found that the most efficient criterion of amplitude is the sum of the R wave in lead 1 and the S wave in lead 3. This sum was less than 25 mm. (2.5 millivolts) in 99% of normal subjects with left axis deviation; whereas, it exceeded 25 mm. in over one-half of cases with left axis deviation in whom there was left ventricular enlargement. Among an unselected group of 380 insurance applicants with hypertension the amplitude of R1 + S3 exceeded 25 mm. in 29.2% of cases. Depression of the ST segment in lead 1 of 0.5 mm. or more, and flattening of the T wave in lead 1 to less than 1 mm. were also found to be signs of left ventricular hypertrophy when associated with left axis deviation, since these changes occurred in large proportion of subjects with hypertrophy of the left ventricle, but were not observed in normal cases. The findings indicate that left ventricular hypertrophy may be considered to be present when left axis deviation is present in association with any of the following abnormalities in the ventricular complex:

1) Increase in amplitude of the Q. R. S. complex, where the sum of the R wave in lead 1 and S wave in lead 3 is over 25 mm. (2.5 millivolts).
2) Depression of the ST segment in lead 1, even of as slight degree as 0.5 mm.
3) Flattening of the T wave below 1 mm. amplitude, or other T wave abnormalities in lead 1.

Applying these electrocardiographic criteria, as well as X-ray evidence of left ventricular hypertrophy and aortic widening, to 100 cases with advanced hypertensive heart disease, it was found that 90% showed some evidence of left ventricular hypertrophy or arteriosclerosis of the aorta or coronary vessels in the electrocardiogram or X-ray. The electrocardiogram was found to provide the most sensitive single criterion for detecting left ventricular hypertrophy.

In order to test the thesis that mortality is higher with

increasing duration of the hypertensive disease, a mortality study according to electrocardiographic findings was carried out in 424 cases of applicants with hypertension. The cases were divided into 4 groups:—

1. Normal electrocardiogram
2. Borderline electrocardiographic changes
3. Pattern of left ventricular hypertrophy
4. Electrocardiographic changes indicative of myocardial disease with or without pattern of hypertrophy

It was found that there was a distinct increase in mortality with progression in the electrocardiogram toward an abnormal pattern, the mortality in the respective groups being 186%, 269%, 344% and 375%. This increase was only in slight part attributable to differences in blood pressure since there was little variation in the different groups. Adjustment for slight increase in the blood pressure still left a marked increase in the mortality ratios in the successive groups. There was a relative homogeneity of body build and age, too, in the different groups, so that the increment in mortality must be considered as correlated with the electrocardiographic findings used as a basis for the classification of the groups.

The results of this preliminary study indicate that the stage of the hypertensive disease, as determined by the electrocardiogram, is an important consideration in the evaluation of insurability. Since the electrocardiogram was normal in only 44% of applicants with hypertension, it would appear worthwhile to have electrocardiograms made routinely in all applicants with hypertension. It is recommended that a joint investigation be undertaken to establish more specifically the influence on mortality of the finding of cardiac hypertrophy and coronary and aortic arteriosclerosis in subjects with hypertension, with the ultimate view of applying the findings in evaluating insurability, in conjunction with other considerations such as the blood pressure.

(The authors are indebted to Messrs. W. J. November, J. P. Mills, and R. A. Porter of the Actuary's Department of The Equitable Life Assurance Society for statistical analyses in connection with this study.)

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PRESIDENT CRAGIN—Thanks very much Dr. Ungerleider.

Dr. William Bolt of the New York Life Insurance Company will open the discussion. Dr. Bolt!

* * * * * *

DR. BOLT—The authors need not apologize for presenting another paper on hypertension. It is one of the most important problems facing us today. A survey of 10,000 records of impairments in insurance cases showed that blood pressure was a factor in 10%. The only condition reported more fre-

quently was build, including overweight and underweight covering 14% of the total. Blood pressure was second with 10% and heart murmurs third with 8½%. It is interesting to look back over the years and note the changes that have taken place in our attitude towards hypertension as our knowledge of it grew and our understanding increased. Some of you will probably remember when blood pressure was first taken in insurance examinations. In 1925, the Joint Committee of the Actuarial Society and this Association published the result of an extensive investigation of blood pressure to be followed by another report at the time of the Medical Impairment Study in 1929. These investigations were based on the simple average of the blood pressure readings at the time of the medical examination without reference to previous findings of high blood pressure or of other impairments.

In 1934, a paper was presented to this Association covering an investigation of hypertension in individuals past forty years of age with special reference to a history of high blood pressure and with due consideration to evidence of pathology in other organs. This paper emphasized the importance of a history of hypertension as well as the effect of other and associated conditions. In 1937, Dr. L. F. MacKenzie and Mr. Pearce Shepherd of the Prudential presented another paper before this Association which in general confirmed these impressions. Underwriters began to realize that there was no more reason to ignore a previous record of hypertension, than there was a previous history of pulmonary tuberculosis or gastric ulcer. One is inclined to doubt, therefore, the suggestion in this paper that underwriting today is based on the average of the highest and the lowest blood pressure readings. If any one is following that practice, he is ignoring the experience of insurance medicine as shown during the past few years. Intermittent elevation of the blood pressure is recognized as the forerunner of hypertensive heart disease both by Clinicians and Medical Directors

alike. This is pointed out, for instance, by Dr. Louis F. Bishop, Jr. in an article on "Soldiers' Heart" in the New York State Journal of Medicine for October, 1941. The authors themselves say that elevation of the blood pressure above normal levels under any circumstances is indicative of hypertension, sooner or later.

Recently, the Joint Committee issued a new blood pressure report based on their 1939 investigation. This study is an advance over previous investigations in many ways, but it is necessary to bear a few facts in mind when applying the results to underwriting. First, actuarial cards in most instances still record the simple average of the blood pressures found on examination without references to previous findings of hypertension or of other impairments. Secondly, one should not be misled because an analysis of a number of cases shows relatively little difference between the simple average and other methods such as the average of the two highest blood pressures. In 5,000 standard policies, 89% had only one reading, 7% had two readings and only 4% had more than two readings. Among 3,350 substandard policies, 52% had one or two readings, 18% had three readings and 30% had more than three readings. The fact that over half the cases, even in the substandard group, had only one or two readings means that the averages mentioned in the report must have been largely affected by the fact that such a great number had only one or two readings. Analysis of the group with more than two readings might have shown a wider spread between, for instance, the average of the two highest blood pressures and the average of all readings. One must remember too that actuarial studies are based on cases that have been accepted for insurance and do not include those that have been eliminated by medical selection. If we allow the simple average of all blood pressures, including the lowest readings, to be the basis of selection, we may grant insurance to many who would otherwise be declined, as it is a simple matter to get enough low readings to offset the high ones.

The comment is also made in the paper that examination of the urine is of little value in hypertension, except to distinguish cases due to renal disease. This may be correct, but is likely to lead to misunderstanding if interpreted, as it may be, that urinalysis is unimportant in hypertensive cases. The 1939 Blood Pressure Study included an investigation of 8,350 cases with so-called "minor impairments". Those with cardiac and renal complications showed a mortality ratio of 21% and 23% respectively, higher than similar groups of hypertension alone. It must be remembered again that this experience does not include all those cases that were declined because of high blood pressure and large amounts of albumin or casts, etc. Furthermore, the main idea of this paper, it seems to me, is to encourage more thorough examination of these hypertensive cases, and a general diagnostic survey certainly includes urinalysis, if only to detect those cases where the kidney is involved.

Hypertension, as the authors point out, may be divided into five major groups. A satisfactory classification is hard to devise because so often it is difficult to determine which comes first, the hypertension or the so-called cause. That hypertensive disease is not simply a matter of high blood pressure, but that there are other associated conditions, may be shown by a study of approximately a thousand records of individuals who have died since the original high blood pressure was reported. Analysis of the records, including conditions found after the high blood pressure was first noted, gives the following result:

High Blood Pressure alone	17%
High Blood Pressure with Cardiac or Renal complications, or both	32%
High Blood Pressure with other single complications	14%
High Blood Pressure with Multiple complications, including Cardiac and Renal	37%

It will be seen that less than 20% showed high blood pressure alone, even granting that some of the other conditions, e. g., nervousness, indigestion, may be symptons associated with the blood pressure. In an appreciable number there is evidence of pathology elsewhere. The important thing from our standpoint is that the more careful the medical examination at the time of application for insurance, the more likely we are to detect other conditions suggestive of long standing hypertension.

One often hears it said that the viewpoint of the Medical Director of a Life Insurance Company is different from that of a Clinician. To some extent that is true. A Medical Director is concerned particularly with prognosis, whereas the Clinician is essentially concerned with the treatment and welfare of his patient. In one thing, however, both are alike. Neither can be successful without an understanding of the underlying pathology in a given case. A good history, a careful examination with the additional help of appropriate special examinations, e. g., x-rays, electrocardiograms and a proper understanding and use of medico-actuarial experience are essential for good underwriting.

X-rays and electrocardiograms are being used more and more in insurance medicine. We have been using them in the New York Life for over ten years. While we have no definite mortality figures to report, we can say that the experience with cases accepted for insurance after these examinations has been satisfactory. There is still a great deal to be learned, however, about the electrocardiogram, and the authors of this paper are to be congratulated on this piece of original work. We need more papers of a similar nature, and efforts in this direction should be encouraged. That does not necessarily mean that when the results of such studies are presented we should not consider them critically. Too many people are inclined to look on the electrocardiogram as a panacea for all our troubles in cardiovascular cases. All the more reason, therefore, for us to be cautious when drawing conclusions from original investigations such as this.

The general criteria for left ventricular strain are well recognized, as may be found in any accepted book on cardiology. Masters, for instance, described the hypertensive electrocardiogram as one with left axis deviation, inversion of T waves, high voltage and displaced ST interval. He emphasizes that the changes are gradual and depend on the duration of the hypertension and degree of left ventricular enlargement, rather than the state of the myocardium. Other investigators have described these changes – left axis deviation, high voltage, depressed ST interval, T wave changes - since 1929 or earlier so that the pattern is now well established.

The authors of this paper have made the commendable effort of trying to establish numerical standards for these changes. They very properly emphasize that left axis deviation is not synonymous with left ventricular hypertrophy, since it is only a term expressing the preponderant electrical axis and is found in other conditions, many of them normal. Furthermore, not every hypertensive case will show the typical electrocardiographic changes because enlargement from hypertrophy of the left ventricle does not always occur. There are cases of long standing hypertension with small hearts in which enlargement is difficult to recognize, possibly because, though there may be a relative increase in the size of the ventricle, the measurements still remain within the limits accepted as normal.

Whether we can be specific in our criteria for the pattern of left ventricular strain is debatable. There is always the question of displacement of the anatomical size of the heart, either horizontally or vertically, and the question is to decide how much of this pattern is due to left ventricular strain and how much to the anatomical axis. The designation of R1 + S3 as the best criterion indicative of voltage is interesting and should stimulate independent studies.

Since receiving this paper, we have rather hurriedly analyzed 200 cases of hypertension, 100 where the individuals have since died heart deaths, and 100 where the individuals are still living, with reference to the measurements of R1 and S3 and the other criteria. In the first group of 100, since dead, there

were 72 which showed left axis deviation and 28 where there was no axis deviation. Of the 72 cases with left axis deviation, R1 + S3 exceeded 25 mm. in 25 cases or 35% and was less than 25 mm. in 47 cases or 65%. Of the 25 cases where R1 and S3 exceeded 25 mm., 19 or 76% had abnormal electrocardiographic findings by ordinary standards (definite T wave changes, RST segmental changes, auricular fibrillation or bundle branch block). Of these 25, 18 were x-rayed, and 14 or 77% showed an enlarged heart or an abnormal aorta or both. Of the 47 cases where R1 + S3 was less than 25 mm., 29 cases or 62% had abnormal electrocardiographic findings. Thirty-eight of the 47 were x-rayed and 23 or 60% showed an enlarged heart, an abnormal aorta or both.

The second group of 100 cases with hypertension, still living, showed left axis deviation in 64 and no axis deviation in 36. In the 64 cases with left axis deviation, R1 + S3 exceeded 25 mm. in 11 cases or 17%, and was less than 25 mm. in 53 cases or 83%. Of the 11 cases where R1 + S3 exceeded 25 mm., 2 cases or 18% had abnormal electrocardiographic findings. Nine of the 11 were x-rayed and 5 or 55% were found to have an enlarged heart, an abnormal aorta or both. Of the 53 cases with R1 + S3 less than 25 mm., 7 or 13% had abnormal elctrocardiograms. Thirty-six of the 53 were x-rayed and 14 or 39% showed an enlarged heart, an abnormal aorta or both.

Out of 200 cases of hypertension then, left axis deviation occurred in 136 or 68%. In this 136, R1 + S3 exceeded 25 mm. in 36 or 26%. A study of these same 136 cases for ST changes showed 25 of the deads and 4 of those still living, or a total of 29, i. e., 21% where the ST changes conformed to the authors' criterion. In the same group, a study of T_1 abnormalities showed 24 cases in the dead and 5 in the living, or a total of 29, i. e., 21% where the T waves in Lead I were abnormal.

The authors say that left axis deviation combined with any one of the three criteria is evidence of left ventricular hyper-

trophy. In this group of 136 with left axis deviation, there were 28 of the dead and 49 of the living or a total of 77, i. e., 57% which showed none of the three criteria. In this same 136, nearly half had abnormal electrocardiograms by usual standards, and approximately half of those that were x-rayed had abnormal findings by our usual standards. Taking the whole group of 200, there were 59 or 30% which had left axis deviation and one of the three criteria, and 141 or 70% which did not present left axis deviation, or having left axis deviation, presented none of the other three criteria. We also studied the 100 deads as a group, feeling that 100 cases of hypertensive disease, which have since died heart deaths, might show greater evidence of these changes because in the group of 100 living there were some where the hypertension was not marked. Of the 100 dead there were 72 with left axis deviation and 28 without left axis deviation. In the 72 with left axis deviation, 44 showed one or more of the other criteria and 28 had none of them. In the whole group of 100 then, there were 44 which conformed to the authors' standards and 56 which did not. An analysis of these 100 cases from the underwriting standpoint may also be interesting. Fifty-six were disability claims, all obviously uninsurable. Nine were examined for reinstatement or something similar. Thirty-five were applicants for new insurance. Only 8 received an offer of any kind and all of the 8 were rated or limited.

These groups, as well as the mortality groups in the paper itself, are much too small to justify any definite conclusions. The figures are merely presented to stimulate further study. One might surmise from these small groups that approximately half of the hypertensive cases might show the authors' criteria for left ventricular hypertrophy. About half too would show abnormalities by the ordinary electrocardiographic and x-ray standards. This applies even to a group like the 100 dead of heart conditions where consideration of all the factors available regarding underwriting would indicate that very few were insurable at the time of the examination.

It is possible that when unfavorable cases are eliminated by careful study including electrocardiograms and x-rays that the rest may be insurable on a relatively favorable basis.

In closing, I want to thank the authors for the privilege of discussing this stimulating paper.

PRESIDENT CRAGIN—We will now call on Dr. Robert M. Daley of the Equitable Life Assurance Society.

DR. DALEY—I have nothing special to contribute, in view of the striking unanimity of the figures, but this study which we have made is the result of studying data which we collected with no particular plan in view. We made certain rules in regard to people who are examined at the home office, not expecting so much value from it, and it is interesting to see the benefit which has come from a study of such data.

Incidentally, this study has been recently concluded, and that is one of the reasons why Dr. Bolt had such short notice, but since that study has been made, we have had two cases with hypertension for noticeable amounts, and have called for an electrocardiogram which we otherwise would not have done, and, unfortunately, they both showed considerable abnormalities, and would have been of value just in the beginning.

PRESIDENT CRAGIN—Thank you, Doctor.

The next speaker I am sure is one whose name is familiar to you all, but he hasn't appeared on our program before as far as I can find out. He has been interested of late in doing some work on the frontal aortic silhouette. Dr. Joyce T. Sheridan is Assistant Medical Director of the Fidelity Mutual Life Insurance Company, and he will now read his paper on "The Transverse Diameter of the Frontal Aortic Silhouette". Dr. Sheridan!

THE TRANSVERSE DIAMETER OF THE FRONTAL AORTIC SILHOUETTE

By Joyce T. Sheridan, M. D.
Fidelity Mutual Life Insurance Company

Increasing use of a single frontal teleoroentgenogram or orthodiagram as a routine procedure in the examination of presumably normal individuals has created a wider need for and extended adoption of more accurate standards of cardiac mensuration. Considerations of time and expense in connection with the examination of applicants for insurance, or routine health examinations, usually preclude more extensive x-ray study in other than the exceptional case. In 1938, Doctors Ungerleider and Clark[1] published criteria as to the correlation between cardiac transverse diameter and build. As this study was based on one thousand, four hundred and sixty teleoroentgenograms of carefully examined normal individuals, it is without doubt the most dependable standard of normalcy now available for this particular measurement. This work, and the prior contributions of Hodges and Eyster[2] and others have largely caused the discard of crude and undependable standards of normalcy such, for instance, as the cardio-thoracic ratio.

The material here presented deals with another measurement, the so called transverse diameter of the frontal aortic silhouette, hereinafter termed the "AA". Preliminary charts covering this study were included in the Scientific Exhibit of The Assocation of Life Insurance Medical Directors in 1939. This measurement should not be confused with the "GV" or transverse measurement of the great vessel shadow at a more or less arbitrary level, as discussed by Dr. Paul White in "Heart Disease".[3] The "AA" is the summation of the distance from the midline to the outer right aortic or great vessel contour and the distance from the midline to the outer left

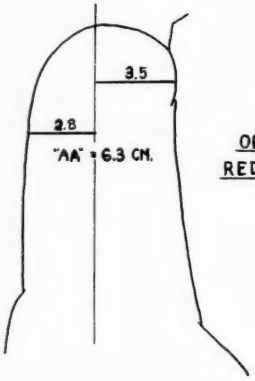


PHOTOGRAPHIC SILHOUETTES



MALE ~ 6' 3½" ~ 243 LBS.

FEMALE ~ 4' 11½" ~ 95½ LBS.



ORTHODIAGRAMS
REDUCED TO SCALE

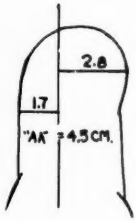
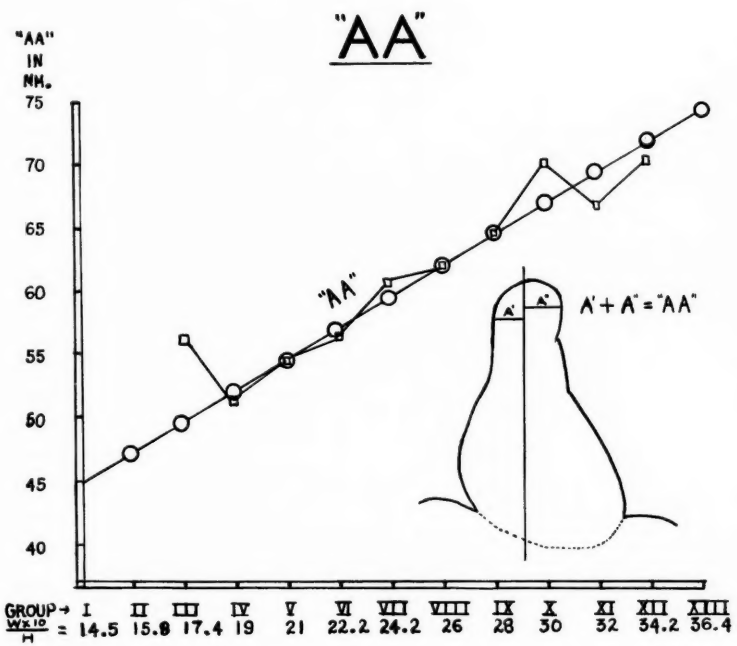


CHART I



Squares Plot Average "AA", uncorrected, found on Orthodiagrams
Circles Plot Corrected "AA", after smoothing Graph
Build Groups are arranged by Clark's Index of Build

CHART II

contour of the descending portion of aortic arch, as illustrated in Chart II. From the anatomical viewpoint this measurement is open to criticism, since the outermost right border may be represented by superior vena cava, aorta, or sternum. Unfortunately, this measurement is the best available in a single frontal film, and if one is to select from the average those cases which demand more extensive study, one must have some knowledge as to what constitutes the average. Furthermore,

from the practical viewpoint, most, if not all of the cases in which the right border is represented by superior vena cava or sternum will have a normal "AA". It may, therefore, be assumed that the right border of any "AA" above normal actually represents the outer right contour of the ascending aorta, when the shadow is not obscured by other pathology. The character of the contour will aid differentiation in the x-ray, whereas characteristic pulsation serves as additional aid fluoroscopically. In any event an "AA" which materially exceeds the average for the patient's build should but serve as an indication for further study, in oblique and lateral positions and including visualization of the aorta with the oesophagus rendered opaque.

It has been rather difficult to trace the origin of the common belief that while aortic size increases quite definitely with age, the influence of build is insignificant. A statement to this effect is found in "The Heart and The Aorta", by Vaquez and Bordet.[4] And in the life insurance field there has been a tendency to accept seven centimeters as the top normal limit for the "AA" without regard to build. This rough guide serves well enough in dealing with median build groups but has undoubtedly misled some who have given to this rough definition of normalcy more significance than was ever intended. Casual observation in fluoroscopy or x-ray mensuration makes it obvious that such a measurement allows a very ample margin in the short underweight and little or no margin in the tall overweight. Chart I illustrates this point. If the aortic silhouette of the small individual is superimposed upon the cardiac silhouette of the large individual, or vice versa, the absurdity of any assumption that aortic size does not vary with build, just as heart size varies with build, becomes strikingly apparent. The two individuals selected for this comparison of cardiac silhouettes, aortic silhouettes and build silhouettes were physically normal in all respects. The larger individual represents an extreme of stature, but not an extreme overweight. The smaller individual approaches the other

extreme of stature, but is not emaciated, being an apparently healthy underweight. Cardiac transverse diameter in each case is very close to the predicted normal, either by criteria of Ungerleider and Clark[1] or by criteria of Hodges and Eyster.[2] The "AA" of each is also within ten per cent of the predicted normal by criteria here presented.

In an effort to translate the mere impressions of observation into usable averages a series of five hundred and twenty-nine orthodiagrams was studied, all made at the Head Office of The Fidelity Mutual Life Insurance Company, in connection with prospective employment, health examinations or applications for insurance. Cases with hypertension, cardiac hypertrophy, evidence of cardiovascular disease or other significant abnormality were eliminated. The orthodiagrams were classified in ten build groups, graduated from the short underweight to the tall overweight. The age of individuals studied ranged from eighteen to sixty-five, the average being forty-three. The build groups were defined by Dr. Clark's index of build, $\frac{W \times 10}{H}$, (Weight multiplied by 10 divided by height), a formula that Dr. Clark[5] found to be very useful in connection with physical characteristics which vary in proportion to total body mass, and the formula used for the build classifications upon which were based Ungerleider and Clark's criteria pertaining to cardiac transverse diameter.

In each of the ten build groups defined, the average "AA" was determined and plotted as shown in Chart II. As will be noted, the average value of the "AA" increases quite regularly as $\frac{W \times 10}{H}$ increases.

In determining averages among relatively small subdivisions of any group, one is hesitant to accept the results as being significant unless findings can be rationalized by comparison with other studies, adequate and sufficiently closely related to justify comparison. As the majority of the orthodiagrams in this study were made by the author, the possible influence of

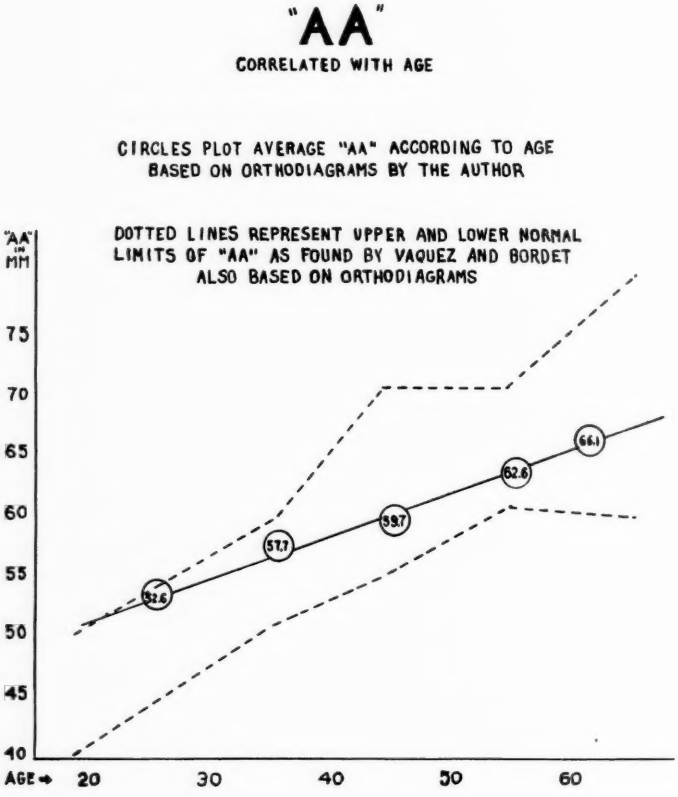


CHART III

the personal equation also made it advisable to reclassify the material that it might be checked against some prior study by independent observers. Vaquez and Bordet in "The Heart and The Aorta"[4] publish their findings of the upper and lower normal limits of the "AA" as related to age, without regard to build. Chart III, therefore, shows the present material re-

classified by age without regard to build, together with the upper and lower normal limits as defined by Vaquez and Bordet. It will be noted that the increase of the "AA" with age is less marked in the present material. This could readily be due to the author's stricter standards of normalcy with regard to hypertension, cardiac hypertrophy, et cetera, particularly at the older ages. In any event, the present data appear to lie sufficiently within the range of normalcy, as defined by these independent observers, to justify use for the immediate purpose.

There is another comparative approach. From the physiological standpoint, it would seem reasonable to expect an interdependent relationship between total body mass, total blood volume, heart size and aortic calibre. It would seem unreasonable for the first three to show such a size and volume relationship without the fourth, the aorta, exhibiting a similar size or volume relationship. If this hypothesis is correct, the average "AA", assuming the "AA" itself varies proportionately with aortic calibre, might be expected to represent a constant proportion of the average "TD" for each build group. In the material reviewed such a constant proportion was found to exist, as is shown in Chart IV. When average values of the "AA" as found by the author were related to the average values of "TD" by criteria of Hodges and Eyster,[2] the "AA" was found to approximate 46% of the "TD", the extreme range being from 44% to 47%. It was further found that the "AA" approximated 45% of the "TD" as determined by Ungerleider and Clark,[1] the extreme range being from 44.6% to 45.5%. In making this comparison with the criteria of Ungerleider and Clark it must be borne in mind that the latter's values of "TD" are based on teleoroentgenograms, whereas the "AA" studies are based on orthodiagrams; also that average values of "TD" teleoroentgenographically by Ungerleider and Clark[1] do not exceed average values of "TD" orthodiagrammatically by Hodges and Eyster[2] by the presumptive eight per cent allowance frequently used in comparing the two techniques.

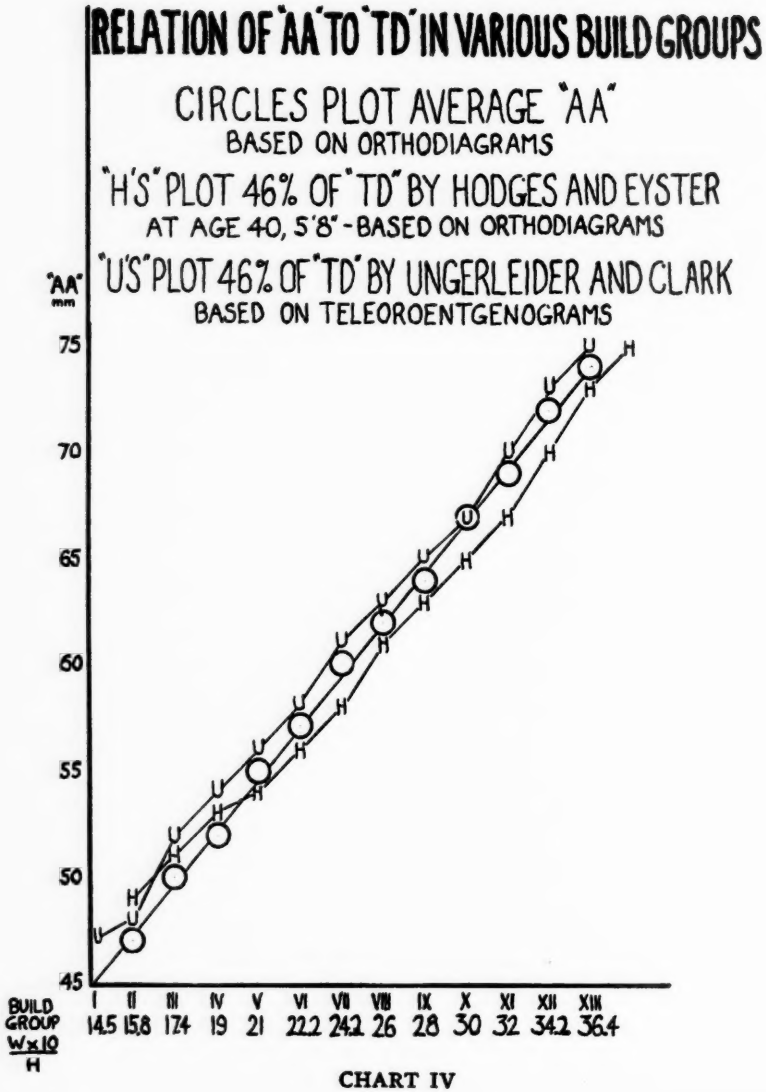

RELATION OF "AA" TO "TD" IN VARIOUS BUILD GROUPS
CIRCLES PLOT AVERAGE "AA"
BASED ON ORTHODIAGRAMS
"H'S" PLOT 46% OF "TD" BY HODGES AND EYSTER
AT AGE 40, 5'8" - BASED ON ORTHODIAGRAMS
"U'S" PLOT 46% OF "TD" BY UNGERLEIDER AND CLARK
BASED ON TELEOROENTGENOGRAMS
"AA" mm
75
70
65
60
55
50
45
BUILD GROUP W x 10 / H
I II III IV V VI VII VIII IX X XI XII XIII
14.5 15.8 17.4 19 21 22.2 24.2 26 28 30 32 34.2 36.4

CHART IV

Considering the volume of material available for this study and the number of build subdivisions necessary, the constancy of the relationship of the average "AA" to the average "TD" is remarkable. So far as the author has been able to determine, this relation of the average "AA" to the average "TD" for various builds has not previously appeared in medical literature. Its practical application is apparent, for in viewing the frontal aortic silhouette one can be guided by the fact that the average transverse diameter of this silhouette bears a known relation to the cardiac transverse diameter for the particular height and weight under consideration. The predicted "AA" may be defined as 46% of the predicted cardiac transverse diameter. For roentgenograms the criteria of Ungerleider and Clark[1] are used to obtain the predicted "TD". For orthodiagrams the data of Hodges and Eyster[2] are used, since several factors appear to make the practice of employing a percentage correction to translate teleoroentgenographic values into equivalent orthodiagraphic values, or vice versa, unreliable. If a correction for age is desired, one millimeter may be subtracted for each three years of age below forty-three and one millimeter added for each three years of age above forty-three. This correction is based on an average age of forty-three and the findings shown in Chart III. One should not, of course, measure the "TD" in a given case and assume the "AA" to be 46% of that "TD", for in dealing with the individual both "AA" and "TD" are variables, the average value of each of which may be predetermined by group study only.

Dr. Hugo Roesler[6] has ably and exhaustively reviewed the general subject of measurements, and it is hardly necessary here to stress the caution which must attend cardiovascular mensuration in a single plane. Like the "TD", the "AA" has certain practical advantages in interpretation of a single frontal cardiovascular silhouette. Even more than the "TD", the "AA" holds pitfalls for misplaced confidence. Two aortas of identical calibre may present different "AA" measurements, by reason of the direction of the arch being more antero-

Theoretical Transverse Diameters of Heart Silhouette and Aortic Silhouette for Various Heights and Weights

"AA" mm.	T. D. of Heart	HEIGHT																				
		5'0"	1"	2"	3"	4"	5"	6"	7"	8"	9"	10"	11"	6'0"	1"	2"	3"	4"	5"	6"		
47	100 mm	83	85	86	87	89	90	92														
	101 "	85	86	88	89	91	92	93	95													
	102 "	87	88	90	91	92	94	95	97													
	103 "	88	90	92	93	94	96	97	99	100												
	104 "	90	92	93	95	96	98	99	101	102												
49	105 "	92	93	95	96	98	99	101	103	104	106											
	106 "	94	95	97	98	100	101	103	104	106	108											
	107 "	95	97	99	100	102	103	105	106	108	110	111										
	108 "	97	99	100	102	104	105	107	108	110	112	113										
	109 "	99	101	102	104	106	107	109	110	112	114	115	117									
52	110 "	101	102	104	106	108	109	111	113	114	116	118	119	121								
	111 "	103	104	106	108	109	111	113	115	116	118	120	121	123	125							
	112 "	105	106	108	110	111	113	115	117	118	120	122	124	125	127	129						
	113 "	106	108	110	112	113	115	117	119	121	123	124	126	128	129	131	133					
	114 "	108	110	112	114	115	117	119	121	123	125	126	128	130	132	133	135	137				
54	115 "	110	112	114	116	117	119	121	123	125	127	129	130	132	134	136	138	140	141			
	116 "	112	114	116	118	120	121	123	125	127	129	131	133	134	136	138	140	142	143	146		
	117 "	114	116	118	120	122	124	125	127	129	131	133	135	137	139	141	143	145	146	148		
	118 "	116	118	120	122	124	126	128	129	131	133	135	137	139	141	143	145	147	149	151		
	119 "	118	120	122	124	126	128	130	132	134	136	138	140	142	143	145	147	149	151	153		
56	120 "	120	122	124	126	128	130	132	134	136	138	140	142	144	146	148	150	152	154	156		
	121 "	122	124	126	128	130	132	134	136	138	140	142	144	146	148	150	152	154	156	159		
	122 "	124	126	128	130	132	134	136	138	140	143	145	147	149	151	153	155	157	159	161		
	123 "	126	128	130	132	134	136	139	141	143	145	147	149	151	153	155	157	160	162	164		
	124 "	128	130	132	134	137	139	141	143	145	147	149	152	154	156	158	160	162	164	166		
58	125 "	130	132	134	137	139	141	143	145	147	150	152	154	156	158	160	163	165	167	169		
	126 "	132	134	137	139	141	143	145	148	150	152	154	156	159	161	163	165	167	170	172		
	127 "	134	137	139	141	143	146	148	150	152	154	157	159	161	163	166	168	170	172	175		
	128 "	136	139	141	143	146	148	150	152	155	157	159	161	164	166	168	171	173	175	177		
	129 "	139	141	143	146	148	150	152	155	157	159	162	164	166	169	171	173	176	178	180		
61	130 "	141	143	145	148	150	152	155	157	160	162	164	167	169	171	174	176	178	181	183		
	131 "	143	145	148	150	152	155	157	160	162	164	167	169	172	174	176	179	181	183	186		
	132 "	145	148	150	152	155	157	160	162	164	167	169	172	174	177	179	181	184	186	189		
	133 "	147	150	152	155	157	160	162	165	167	169	172	174	177	179	182	184	187	189	192		
	134 "	150	152	155	157	160	162	164	167	169	172	174	177	179	182	184	187	189	192	194		
63	135 "	152	154	157	159	162	164	167	169	172	175	177	180	182	185	187	190	192	195	197		
	136 "	154	157	159	162	164	167	169	172	175	177	180	182	185	187	190	193	195	198	200		
	137 "	156	159	162	164	167	169	172	175	177	180	182	185	188	190	193	195	198	201	203		
	138 "	159	161	164	167	169	172	174	177	180	182	185	188	190	193	196	198	201	204	206		
	139 "	161	164	166	169	172	174	177	180	182	185	188	190	193	196	198	201	204	206	209		
65	140 "	163	166	169	171	174	177	180	182	185	188	190	193	196	199	201	204	207	209	212		
	141 "	166	168	171	174	177	179	182	185	188	190	193	196	199	201	204	207	210	212	215		
	142 "	168	171	174	176	179	182	185	188	190	193	196	199	202	204	207	210	213	216	218		
	143 "	170	173	176	179	182	184	187	190	193	196	199	202	204	207	210	213	216	219	221		
	144 "	173	176	178	181	184	187	190	193	196	199	201	204	207	210	213	216	219	222	224		
67	145 "	175	178	181	184	187	190	193	196	198	201	204	207	210	213	216	219	222	225	228		
	146 "	178	180	183	186	189	192	195	198	201	204	207	210	213	216	219	222	225	228	231		
	147 "	180	183	186	189	192	195	198	201	204	207	210	213	216	219	222	225	228	231	234		
	148 "	182	185	188	192	195	198	201	204	207	210	213	216	219	222	225	228	231	234	237		
	149 "	185	188	191	194	197	200	203	206	210	213	216	219	222	225	228	231	234	237	240		
70	150 "	187	191	194	197	200	203	206	209	212	215	219	222	225	228	231	234	237	240	243		
	151 "	190	193	196	199	203	206	209	212	215	218	221	225	228	231	234	237	241	244	247		
	152 "	192	196	199	202	205	208	212	215	218	221	224	228	231	234	237	240	244	247	250		
	153 "	195	198	201	205	208	211	214	218	221	224	227	231	234	237	240	244	247	250	253		
	154 "	198	201	204	207	211	214	217	221	224	227	230	234	237	240	244	247	250	253	257		
73	155 "	200	203	207	210	213	217	220	224	227	230	233	237	240	243	247	250	253	257	260		
	156 "		206	210	213	216	220	223	227	230	233	236	240	243	247	250	254	257	260	264		
	157 "				216	219	222	226	229	233	236	239	243	246	250	253	257	260	263	267		
	158 "						225	229	232	236	239	243	246	249	253	256	260	263	267	270		
	159 "								235	239	242	246	249	253	256	260	263	267	270	274		
75	160 "									245	249	252	256	259	263	266	270	274	277			
	161 "											255	259	263	266	270	273	277	281			
	162 "											259	262	266	270	273	277	280	284			
	163 "													269	273	276	280	284	287			
	164 "													273	276	280	284	287	291			

Refer to paper entitled "A Study of the Transverse Diameter of the Heart Silhouette with Prediction Table Based on the Teleroentgenogram" presented to the Association of Life Insurance Medical Directors of America by Dr. Henry E. Ungerleider of the Equitable Life Assurance Society, and Dr. Charles P. Clark, of the Mutual Benefit Life Insurance Company, 1938, and to paper entitled "The Transverse Diameter of the Frontal Aortic Silhouette" presented to the same Association by Dr. Joyce T. Sheridan, of the Fidelity Mutual Life Insurance Company, 1941

CHART V

posterior, or more transverse, or for other anatomical reasons independent of aortic abnormality. Consequently, an abnormally large "AA" should serve merely as indication for further study of the region of the aortic silhouette in all planes and also with the oesophagus rendered opaque. This is a task for one thoroughly versed in roentgenological cardiovascular diagnosis.

Conversely, the shortcomings of the "AA" as a measurement should not lead to its disregard. Lack of knowledge as to what constitutes an average value of this measurement may in itself be responsible for neglect of indicated study, with consequent failure to recognize significant pathology.

Obviously a preliminary study of this scope can hardly establish exact values. It is presented, however, in the hope that this demonstration of the influence of build on the transverse measurement of the aortic silhouette will stimulate further similar correlations by others, preferably with survey of a sufficiently large volume of material to permit subdivision by both age and build. Meanwhile, interpretation of the frontal orthodiagrams and teleoroentgenograms may be aided by the present finding that the average transverse diameter of the frontal aortic silhouette approximates forty-six percent of the average cardiac transverse diameter for any given height and weight. For convenience, the original table of "Theoretical Transverse Diameters of Heart Silhouette for Various Heights and Weights" by Doctors Ungerleider and Clark, is here reproduced as Chart V, with an added column from which the average "AA" for build, so computed, may be read directly.

SUMMARY

1. A study of orthodiagrams of normal adults leads to the conclusion that the transverse diameter of the aortic silhouette in the frontal plane, herein termed the "AA", increases as the value of Dr. Clark's index of build, $\frac{W \times 10}{H}$, increases.

2. The average transverse diameter of the frontal aortic silhouette, "AA", is shown to bear a constant relationship to the average cardiac transverse diameter, "TD", for any given build.

3. It appears that the average "AA" for any build may be defined as forty-six percent of the average "TD" for the same build.

4. Values of the transverse diameter of the frontal aortic silhouette, so computed, have been incorporated as column "AA" in Doctors Ungerleider and Clark's table of Cardiac Transverse Diameters, here reproduced.

5. Subtraction of 1 mm. for each three years of age below forty-three or addition of 1 mm. for each three years above forty-three appears to serve as a suitable age correction of the "AA" for adults.

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PRESIDENT CRAGIN—Thank you very much Dr. Sheridan for your interesting summary, and we look forward to reviewing the complete paper in the transactions. The subject will be discussed by Dr. H. H. Fellows of the Metropolitan. Dr. Fellows!

DR. FELLOWS—Before reporting on the notes which I have here, I want to acknowledge the painstaking and detailed work that was done by our Medical Statisticians, Mrs. Woods and Mrs. Stephens. They have been of inestimable assistance throughout this study.

Together in 1936 we set up a plan of study, using the annual examinations of our employee group as a means of obtaining data. First we defined our criteria of normalcy, which was that there must not be the slightest question of cardiac impairment of anyone in the normal group. There must be no history of hypertension (and our limits were 140 systolic and 90 diastolic); there must be no history of heart impairment; there must be no abnormal heart finding at the time that the annual examination was made, and there must be no asterisk or plus codes on the routine annual electrocardiogram. The abnormals were those who could not qualify as normals.

There were 1,722 Home Office employees over age 40 in this group, all of whom had had one or more x-ray examination of the heart and the aorta, but let's deal in round numbers, as they are easier to follow. The distribution by sex was just about the way we would like to have it, nearly half and half, 900 males and 800 females. The age distribution in these 1,700 was evenly distributed from 40 to age 60, but since our female employees may retire at age 60, there were fewer females past age 60 and proportionately more males.

Six hundred of our 1,700 Home Office employees over age 40 were normal by the foregoing criteria, leaving 1,100 that were abnormal. The age and sex distribution in the normal and abnormal groups was exactly the same as it was in the entire group.

And now we come to the small part which I had in this work. I was given the posteroanterior and oblique x-rays of

the thorax and a card which I was told to fill out. The statisticians said, "Don't look at anything except the x-ray and the card. Measure the transverse diameter of the heart and measure the aorta in certain places, and then we will work out correlations with the other data." And they did a beautiful job.

Sheridan, working with a group that were normal by his definitions, made measurements of the aorta and prepared a table which you will see in the proceedings. We, having perhaps an even more strict definition and using medical information about our group of normals through annual examinations going back twenty years, find that we agree entirely with everything Sheridan has said. We found that the aortic measurements of our normals were always within five percent of Sheridan's in each age and weight group, and in most groups it was closer than five per cent.

We found also if we place all the cases in our group which are over plus 10 percent of the normal Sheridan Prediction Table, that only 16 percent of our normals fall in that group while 84 percent of all of the cases over plus 10 are abnormal, having one or more of the following impairments; increased tension, electrocardiographic codes that are unfavorable, a history of cardiac disease, or a murmur. In other words, if you are confronted with the measurements of five aortas that are more than plus 10 percent of Sheridan's Prediction Table, the odds are about four to one that you are dealing with an abnormal person, and you may expect to find additional impairments. If any of you like the odds of four to one against you, that is all right with me.

So, we agree with Sheridan's measurements, with his conclusions, and have had an opportunity to go one step further and say that if you have individuals whose aortas are greater than plus 10 per cent of the normal prediction figure, the odds are at least four to one that you are dealing with a person who has other related impairments which are associated with cardiac abnormality.

Now, this is where I may be supposed to stop, but I am

not going to do it because this job that we have been working on is going to give a lot of comfort, although they don't need it, to both Doctors Ungerleider and Clark. In addition to making an aortic measurement, we measured the hearts in our group, tested them against the Ungerleider and Clark normal heart prediction table, and I never saw such an amazing result. Ungerleider and Clark worked with one group at one time, Sheridan worked in Philadelphia on a different group of individuals, and we worked in New York, and all report independently the same results.

The measurements of the hearts in our group show that our normals are within five percent of the average prediction value that Ungerleider and Clark arrived at in 1938. Of all persons with transverse cardiac measurements greater than plus 10 percent, there were only 18 percent of the normals as against 82 percent of the abnormals. So again the odds are about the same. In our observations it didn't make any difference, then, whether we measured the heart or the aorta in an x-ray, if it is more than plus 10 percent of average, the odds are strongly against the probability that you are dealing with a normal individual.

I have talked to some skeptics about these prediction tables because frequently one gets more help from people who don't agree with you than from those who do. There is one of my friends who just doesn't believe that these tables are worth while and in talking with him, he gave me a hint that was worth following. After talking with him we dug out of our files the records of applicants for insurance examined at the Home Office, whose records we have been following, and who had died a cardiac death.

You may remember that we have been following the course of certain applicants for insurance examined at the Home Office, whether they are rejected or accepted. There have not been many deaths that could be called strictly cardiac deaths in that group, but we had over a hundred, perhaps enough to give us a trend. Again the Medical Statisticians

gave me the x-rays and said, "Go measure them and we will correlate the measurements and other data and tell you what they show." So they were measured and these are the results. Among the applicants examined at the Home Office who had no insurance history, whose physical examination was entirely normal, we had 35 deaths, just a small group, but if we had had an Ungerleider-Clark Heart Prediction Table and had tried to use it, we would have found this, that 20 percent of these applicants with no insurance history and no abnormal physical findings who had died a cardiac death, had a heart size of over plus 10 percent.

In the next group, those who had an M. I. B. record which made it necessary to bring them to our Home Office for examination, but whose Home Office examination did not confirm that record, 36 percent of the cardiac deaths were in persons whose heart measured more than 10 pecent over the average normal.

In the cases that were rated or declined on the basis of present or previous adverse findings, 49 percent of those who died a cardiac death had a heart size more than plus 10 percent.

In summarizing this part of the discussion, let's think back for just a minute. In 1933, three members of this Association, Doctors Nichols, Turner and Ungerleider read a paper wherein they recommended that we accept the work of Hodges and Eyster, which had not been publicized widely, as a recommended standard for heart size. As far as I know, that was the first time that any large group of physicians agreed to attempt to determine whether normal or average heart size could be determined in relation to body build, age, and weight. That was the preliminary paper. In 1938, Ungerleider and Clark presented to you a Prediction Table, which I suppose is not known to many of the physicians in this country, yet it is a magnificent product.

The next contribution you have heard today. Sheridan has brought his work, begun in about 1938, up to date, and in 1941

we have here two tables with values which can, I believe, be accepted with confidence.

PRESIDENT CRAGIN—Thank you very much Dr. Fellows.

Discussion will now be continued by that mathematical shark of the Mutual Benefit, Dr. Charles B. Clark.

DR. CLARK—It was with pleasure that I accepted Dr. Sheridan's invitation to discuss his Study of the Transverse Diameter of the Frontal Aortic Silhouette. The chief purpose for its presentation to our Society was, Dr. Sheridan states, that it would stimulate further research on this subject. I am sure that we have all enjoyed the report of the study made by Dr. Fellows which adds materially to the statements given by Dr. Sheridan in his own paper, and I am happy, indeed, to present the results of my own examination of the material which I have at hand.

When Dr. Ungerleider presented to me the cardiac and other measurements of approximately 2,200 individuals examined at the Home Office of the Equitable Life Assurance Society, he fortunately included on each card the transverse diameter of the aortic silhouette. It has been possible, therefore, for me to make a study of these cards individually in order to determine what percentage relationship such diameter bears to the theoretical transverse diameter of the heart as given for the corresponding height and weight in the table published by us in 1938. The question may naturally arise—why did we not ourselves include in our original paper a study of the transverse diameter of the aortic arch silhouette since the material was available? Our reasons for not so doing were:

First: a preliminary survey of the material at hand indicated a much greater deviation from the average in the aortic group than in the cardiac group. The reason for this will be demonstrated later.

The second reason was that Dr. Ungerleider, who was responsible for the interpretation of the original chest plates felt very strongly that there is no clear cut method for de-

termining the right and left borders of the aortic silhouette for all cases, since there is a fairly large group in which the right border distinctly cannot be ascertained. Furthermore, in many instances, a density located where one would expect the right aortic border to be present cannot always be proved to be due to the ascending aorta. In other words, one does not get a clean-cut picture that one receives in a study of the films with respect to the transverse diameter of the heart in which the right and left borders can in most cases be determined with reasonable accuracy.

I was quite pleased, nevertheless, to learn from Dr. Sheridan that he had made a study based on a large number of orthodiagrams and had arrived at a reasonable method for determining the percentage relationship of the aortic silhouette diameter with expected transverse diameter of the heart for heights and weights as determined by the criteria of Hodges and Eyster based also upon orthodiagram studies.

The method pursued by me in the present study of the material presented by Dr. Ungerleider was to determine for each of the 1,460 normals, the percentage of the transverse diameter of the aortic silhouette in relation to the theoretical heart diameter as given in the Ungerleider-Clark table. It was found that the greatest number of cases fell at two points, namely at 44% and at 48%. We assumed, therefore, that 46% might be considered the zero deviation point, and we plotted a curve accordingly, on the assumption that the average transverse diameter of the aortic silhouette is 46% of the theoretical transverse diameter of the heart. The plot of the whole material gives a picture which is considerably different from the distribution curve which we secured in our study of the transverse diameters of the heart:

In the first place, the peak is apparently much lower in the curve of the aortic silhouette diameters than is that of the cardiac diameters.

Second, the peak is not as sharp and the spread is much greater.

Third, the distribution curve is quite symmetrical from −25% to +25% deviations, and it is an interesting fact that 624 cards fell between −25% to zero, and 630 from +25% to zero (one-half the zero deviation cards being added to each group).

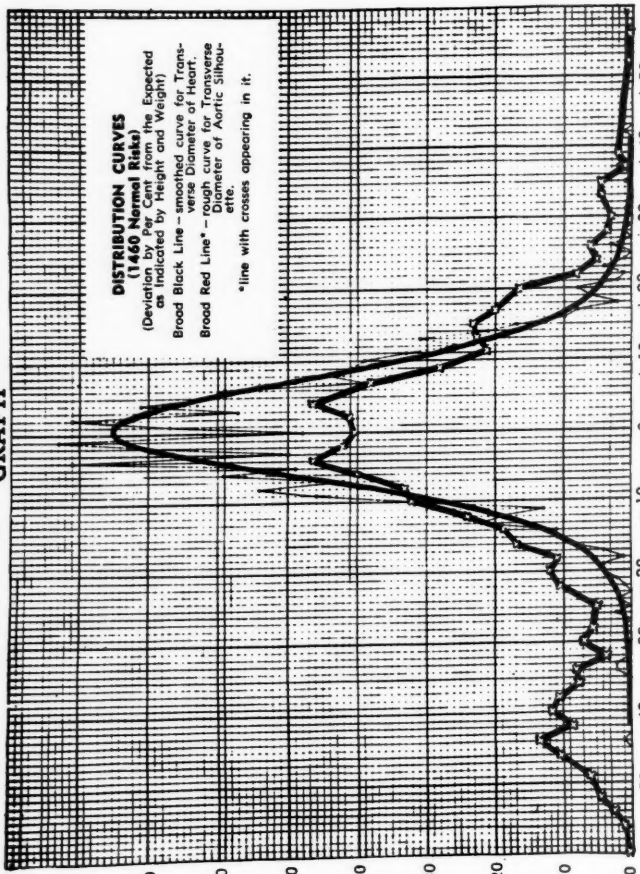
Fourth, a very important difference between the two curves lies in the groups below −25% and above +25% deviation for in our study of transverse diameter of the heart, an equal number fell on the minus side and on the plus side. In the aortic silhouette study, 15% of our 1,460 cases were below the minus 25% deviation, whereas only 3% were above the plus 25% deviation point.

It is evident that the reason for such a large group falling below −25% is that the right border (or in some the left border) often merged with the shadows of the sternum, pulmonary densities, etc., and the exact transverse diameter of the silhouette could not be determined. (Dr. Ungerleider tells me that many of these aortic measurements were only from midsternal line to the margin of the aortic knob.)

I do not wish, myself, to attempt to discuss the various difficulties in interpretation of the aortic silhouette, for Dr. Sheridan and Dr. Fellows are better qualified than I to discuss this particular point.

I am convinced, however, that after my study of the figures based on teleoroentgenograms made by Dr. Ungerleider, we can agree with Dr. Sheridan that the theoretical transverse diameter of the aortic arch silhouette may be assumed to be 46% of the transverse diameter of the heart. This decision is based not upon the average transverse diameter of the aortic silhouette, but upon the frequency as determined by the distribution curve. (See Graph.) I, therefore, am willing to endorse the figures given by him in the column marked "AA" in his Chart 5. It must, of course, be assumed that the observer has followed the same technique for measurement as that employed by Dr. Sheridan and Dr. Ungerleider.

GRAPH


DISTRIBUTION CURVES
(1460 Normal Risks)
(Deviation by Per Cent from the Expected as Indicated by Height and Weight)
Broad Black Line – smoothed curve for Transverse Diameter of Heart.
Broad Red Line* – rough curve for Transverse Diameter of Aortic Silhouette.
*line with crosses appearing in it.
Number of cases
80
70
60
50
40
30
20
10
0
– 50
– 40
– 30
– 20
– 10
0
+ 10
+ 20
+ 30
+ 40
+ 50
Percentage Deviations

Only one other point do I wish to discuss at this time, and that is the correlation between the systolic blood pressure and the transverse diameter under discussion. Because of the large number of cases falling in the group below –25%, it is not possible to determine the average number of millimeters above or below the expected for various groups of blood pressure levels. A significant study, however, is possible, and that I shall give.

There were 835 cases in which the systolic blood pressure was below 130 mm. Of this group, 58% had transverse diameters which were less than the estimated in Dr. Sheridan's table. Thirty-seven per cent were greater than the estimated. Contrasted with this finding, were 493 cards in which the systolic pressure was above 150 mm. Of this, 36% were below the expected and 60% were above. It is evident, therefore, that there is a distinct correlation between blood pressure level and the width of the aortic silhouette.

There is a general feeling, I believe, by most, if not all of our insurance companies, that when the transverse diameter of the heart exceeds +10% of the theoretical, we should suspect that a true cardiac hypertrophy is present. In view of the greater difficulty in accurately measuring the transverse diameter of the aortic silhouette and the greater spread as indicated by the distribution curve, one might well hesitate to assume that a deviation beyond +10% of the expected is necessarily indicative of a true aortic dilatation. However, we are all, I am sure, greatly impressed by the statement of Dr. Fellows that a study of the cases showing a deviation beyond +10% shows there is definite evidence of cardiovascular disease in four of every five cases. Consequently, I am sure that we should regard +10% as the limit of acceptability for standard insurance. Furthermore, such statement by Dr. Fellows leads us to place more confidence in the accuracy of the figures given by Dr. Sheridan as an amendment to the table published by Dr. Ungerleider and myself.

I cannot close this discussion without calling attention to the fact that although Dr. Sheridan, Dr. Fellows and I, working with entirely different material, agree absolutely as the result of mathematical studies, that the transverse diameter of the aortic heart silhouette is theoretically 46% of the transverse diameter of the cardiac silhouette. A further remarkable agreement was shown by our studies in that, having made such an assumption, each of us found that 16% to 17% of our normal cases were beyond the +10% limit.

PRESIDENT CRAGIN—Thank you very much Dr. Clark. Dr. Sheridan, will you close, please?

DR. SHERIDAN—Gentlemen, Dr. Fellows just called my attention to a little item in his notes. It so happens that he had recorded his original study of transverse diameters of the aorta silhouette, and if one uses that group of his cases which show the same blood pressure pictures as those of Dr. Clark, the two are almost in exact agreement.

Is that correct, Dr. Fellows?

DR. FELLOWS—Yes. Dr. Clark simply said using the group that had a little higher pressure than the 140 over 90. We had additional data on that additional group, and taking the group with five millimeters more of increased blood pressure in our material, ours were identical with his again. In other words, we had it figured on that same basis, but we didn't know he was going to use that criterion. In other words, we checked with him again on that.

DR. SHERIDAN—There was one other point that arose in Dr. Ungerleider's mention of the aortic frontal diameter. You may have noted that Dr. Ungerleider used the term "Vascular pedicle" instead of transverse diameter of the frontal aortic silhouette. This is an important point, because one of my great difficulties in putting this work on paper was the question of terminology.

Anatomically, in individuals under forty years of age, the right border is more frequently represented by the superior vena cava, whereas in the age group with which we deal in

insurance, at ages of forty and beyond, the right border, according to Dr. Paul White of Boston, Dr. Kurtz of Wisconsin, and according to most of the men who have looked at the aorta clinically and had not looked at the shrunken, inelastic, no longer pulsating, no longer pulse spread of the dead man, almost all these clinicians agree that in men beyond forty, when we look at the right border we usually see the border of the ascending aorta. For that reason, if we wish to be meticulously accurate in our anatomical terminology in reference to the transverse diameter of the frontal aortic silhouette, we perhaps should not always say this equals a certain number of centimeters, but we should rather say that the transverse diameter of the frontal aortic silhouette is equal to or less than the measurement we can actually make in a frontal film. That completely removes the argument that has long existed between the anatomists and the clinicians on attempting to make this particular measure.

Another thing is this: While I have found that this measurement has helped us in our work, I think there is a certain danger in considering a wide diameter as a convincing proof of aortic dilatation. The measurement may be widened in some places by a curvature of the spine, which throws out the descending aorta slightly.

Now, I think we can say this much, that so far as the single frontal film is concerned, we should consider any case with a definitely wide measurement as a pathological case until that case has been studied in the oblique position and with the aorta visualized by rendering the esophagus opaque with barium, a very accurate measurement.

Those cases will be a very small percentage of the total volume of cases that we handle in our cases and we naturally cannot afford from the economic standpoint the complete examination as a matter of routine.

We have received, I think, from Dr. Fellows, Dr. Clark, and Dr. Ungerleider, not a discussion of this subject but a magnificent contribution. I wonder if we realize how many

hours of work have been boiled down into less than two hours this morning. Hodges and Eyster in 1928 published their criteria on only 80 cases. Dr. Clark has drawn his normals from a careful study of a group of over 2,000 cases on cardiac transverse diameter. Dr. Fellows has drawn his group of normals from complete examinations of 1,720 individuals, and to me, it is astounding that one medical body could have contributed so much in so short a space of time when we consider that the entire field of medicine over a period of ten years added very little to Hodges and Eyster's original work on 80 cases.

Dr. Fellows' correlation is naturally encouraging and satisfying to me because of the startling agreement between his transverse diameters and the diameters on the smaller and less extensive study.

There is one other thing that I want to call attention to before we close this subject, and that is the fact that it was Dr. Clark's early work in build studies that has laid the very foundation of build groupings on which these correlations which we have discussed have been based.

PRESIDENT CRAGIN—The papers that have been read this morning and discussed so thoroughly I am sure have added to our knowledge and I want to thank again the men who have so kindly given their time and their efforts to the work.

Gentlemen, we have had a great many papers in the past on different stomach conditions, more particularly on the question of ulcers and hyperacidity, so it seems to be fitting at this time that the question of achlorhydria should be considered.

Our next speaker holds an eminent position in the field of gastro-enterology, being the author of textbooks and numerous articles on "Affections of the Stomach." He is a gastro-enterologist at Mt. Sinai Hospital, Consulting Enterologist of St. Joseph's, Rockaway Beach, Beth Israel, Newark, New Jersey, United States Veterans' Bureau and the Federal Trade Commis-

sion, a member of the American Gastro-enterology Society, the New York Academy of Medicine, the Society of Experimental Medicine and Biology, the Harvey Society, the International Congress of Gastro-enterologists.

He has appeared before this Association before in discussing factors in selection of risks with a history of gall bladder diseases.

I present Dr. Burrill B. Crohn, who will speak on "Achlorhydria: Its Ultimate Significance."

DR. CROHN—Mr. Chairman, Ladies and Gentlemen: I am duly appreciative of the honor of being asked to address this Association for the second time, and would like to thank you for the privilege. I presume by this time you know I am skeptical of gastritis or the significance of gastritis, and I am skeptical about the findings and the readings of the gastroscope when it is used for purposes for the discovery of organic processes, particularly in the interpretation of the finer changes which are called by so many different observers, gastritis.

ACHLORHYDRIA: ITS ULTIMATE SIGNIFICANCE

By Burrill B. Crohn, M. D.

Achlorhydria, signifying the absence of free hydrochloric acid in the gastric secretion, is a condition rather than a disease. That it regularly occurs as a so-called normal incident in a varied percentage of the controlled population would indicate the fact that its very presence is without clinical significance or actuarial importance. On the other hand, achlorhydria is commonly associated with various diseases as an incidental finding or associated in a clinical sense; by this very coexistence, be it causal or induced, it assumes note-worthy importance.

In the past, some confusion arose due to multiplicity of terms and multiplicity of methods for the determination of its existence. Achlorhydria and anacidity are synonymous as indicating absence of free hydrochloric acid; in most of these instances a small amount of total or combined acid is present; but since pepsin is active only in the presence of free hydrochloric acid, gastric digestion of proteins is suspended under anacid conditions.

Not sufficient attention has been paid to achylia gastrica, a status in which there is absence of free acid, an absolute minimum of total acidity, a reduction of the actual volume of secretion to a few c. c. of mucoid neutral matter, and a complete absence of the ferments pepsin and rennin. This extreme end-stage of the process is differentiated only in degree from simple achlorhydria; achylia however, is very significant because of its close relationship to one condition only, namely, pernicious anemia.

Ewald test-meals were used in the long past; the fractional or Rehfuss meal with oatmeal or alcohol is the method of the present. Where anacidity occurs, an injection of histamine

intramuscularly, in doses ranging from 1/4 to 1/2 mgm. usually provokes some free acid, except in those cases of true achylia gastrica. The insufficiently controlled methods of the past have invalidated to a certain extent, many of the statistical tables on the occurrence of achlorhydria in the larger groups of population when studied. Achlorhydria with an Ewald test-meal may very well be a spurious achlorhydria; by means of a prolonged Rehfuss meal, a large percentage of achlorhydria cases will be eliminated particularly when histamine is used as a test substance. In general however, achlorhydria is significant by all three methods even though we may scientifically differentiate the changes and the degrees.

In any decade of life a percentage of the normal population bears an achlorhydria; this percentage which begins in childhood at perhaps 4 years, (Wright) (1) increases with each decade up to the age of perhaps 60; thereafter it falls off slightly in a diminuendo. The range of anacidity in the so-called normal population, according to Vanzant and her co-workers (2), ranges in males, from 3.6% in the second decade with almost a constant increase to a maximum of 23.1%; the corresponding figures in females begins at 3.2% and reaches as high as 28.1% between the ages of 60 and 64. It is of biological interest to note that acidity is higher in man, a significant factor in understanding the greater increase of duodenal ulcer in males; the incidence of achylorhydria is greater in females in all decades of life.

It might be of interest to pause and ponder over the significance of the existence of so wide-spread an anacidity in so large a group of the controlled population devoid of gastro-intestinal symptoms. Why the achlorhydria? There is no satisfactory explanation. If it were an abiotrophic phenomenon, one would hardly expect to find it in children, in some cases even in the new-born. We would certainly not expect to find it as early as the third decade of life, nor could one expect the falling off of incidence after the 6th or 7th decade.

Can one presume to say that this is a progressive gastritis? Very few observers will agree today with Faber (3) and his

SPRUE—BLOOD PICTURE and GASTRIC SECRETION

SPRUE - BLOOD PICTURE and GASTRIC SECRETION

CASE	%HEMOGLOBIN	RED BLOOD COUNT	COLOR INDEX	REHFUS TEST FREE ACID
1	14	820,000	.9	40
2	65	3,500,000	.9	0
3	88	5,100,000	.9	10
4	22	1,100,000	1.0	40
5	17	750,000	1.1	0
6	25	1,200,000	1.1	42
7	30	1,100,000	1.1	0
8	60	2,400,000	1.1	30
9	63	2,600,000	1.1	10
10	33	1,440,000	1.2	34
11	32	1,420,000	1.2	50
12	82	3,400,000	1.2	0
13	90	4,100,000	1.2	28
14	51	1,830,000	1.3	40
15	51	2,000,000	1.3	52
16	60	2,400,000	1.3	46
17	70	2,800,000	1.3	0
18	65	2,200,000	1.4	44

SUMMARY:

HISTAMINE ANACIDITY - 5 cases
HYPOACIDITY - 2 "
NORMAL ACIDITY - 11 "

A.

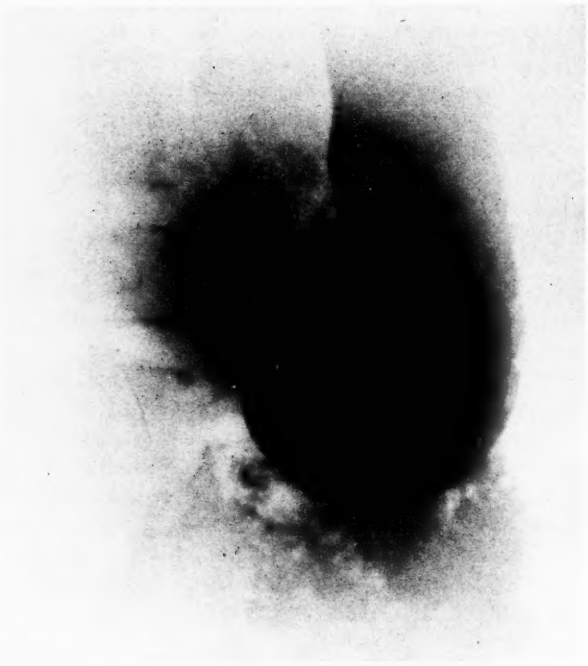
Color Index in Sprue. Note that the color index and the hemoglobin percentage is not directly related to the degree of gastric acidity or anacidity.

school, who have held that gastritis is an organic progressive disease associated particularly with infections such as nephritis, diabetes, tuberculosis and diphtheria; or as a condition related to rapid eating, bad dentures, a poor choice of foods, or the abuse of alcohol and tobacco. Nor for that matter is chronic gastritis an explanation of progressive anacidity to any observers, except perhaps Schindler. For there has been established no standard for the histological study of the mucosa of the normal stomach; nor is it possible to believe that an interstitial cirrhosis or inflammation of the gastric wall is sufficient in degree or in kind to inhibit acid secretion in so large a percentage of the otherwise asymptomatic population. Round cells, interstitial cells, and plasma cells and increase of connective tissues can be observed in amost all cut speci-

mens of stomach at almost all ages. Such changes have never been consistently associated with acidity variations. Nor is the gastroscopic picture as seen through the flexible instrument consistent with the various titrations of acidity. Not alone is the gastroscopic picture of gastritis variable and often doubtful of significance, but even if one were to grant that the gastroscopic picture is that of a hypertrophic, or an atrophic, or a superficial gastritis, in the overwhelming percentage of cases one cannot predict what the acidity finding will be. Only the most advanced achylia may be consistent with a true anadenie or atrophy of the mucosa such as is seen in a pernicious anemia. And even that picture has exceptions.

Gaither and Borlund (4) made careful gastropic studies in achylia gastrica and found no consistent mucosal changes that were consistent with the absence of acid. Even more interesting was the experience of Schiff (5) who, over the course of years on the same willing subject, injected histamine once or twice daily, withdrawing the acid secretion from the stomach of the patient under examination. After a few months of this daily experiment, an anacidity was created in the stomach of this man, which anacidity persisted for a considerable time. With the cessation of the histamine injections; the gastric acidity gradually returned; with the reintroduction of the histamine, another period of acid exhaustion was observed. Gastroscopic studies carried on throughout this extended period of observation failed to show changes in the mucosa of any kind, and certainly nothing that had any significance as regards secretion or lack of secretion.

Years ago Einhorn (6) pointed out that a simple achylia gastrica with complete absence of acid and ferments might co-exist with a normal gastric mucosa, and I, personally, have had experiences which confirm this statement. Certainly in the ordinary everyday achlorhydria, as found in 5% of the controlled population, the histological changes fail completely to explain the incidence of the anacidity. One should



B.

Apparent antral defect in a case of so-called antral gastritis with spasm.

probably look for the explanation outside of the stomach itself; perhaps in the nervous control, perhaps in the lack of production of a secretory hormone such as "gastrin" found and suggested by Starling (7), even though a true specific gastric hormone controlling secretions has been questioned by all subsequent workers.

After subtotal gastrectomy one frequently sees complete anacidity with a perfectly intact fundal mucosa, with chief

and acid cells perfectly preserved; yet no acid juice is being secreted. Perhaps inhibitory substances, such as the enterogastrone of Ivy (8), or urogastrone as found in urine, can under apparently normal conditions inhibit gastric secretion to the point of anacidity.

Martius (9) is the exponent of the constitutional theory of achlorhydria; he emphasizes the not infrequent familial incidence of achlorhydria and the tendency to find numerous cases of achlorhydria in various generations or parallel branches of the same family, a fact confirmed by Mustelin (10), Hurst (11), and others. The constitutional or familial incidence of achlorhydria may be assumed, but it does not explain why one finds in functional disturbances such as the neuroses, constipation, anorexia, and the many subjective psychoneurotic manifestations, a percentage of achlorhydria. Here the achlorhydria is probably secondary to the general nervous manifestations, for achlorhydria, in itself, gives no primary symptoms.

Anacidity is truly asymptomatic, except that classical medicine has always taught that achlorhydria causes diarrhea; one speaks of gastrogenous diarrhea as if it were a common occurrence. Actually, probably less than 5% of the cases of true achlorhydria exhibit diarrhea; most of the subjects of anacidity are constipated or have normal bowel movements, the digestion is good, the intestinal and pancreatic secretions are ample, taking over the function of the stomach in the digestion of proteins and lipoids. Constitutional or metabolic disturbances depending presumably on achlorhydria do not exist.

Even radiographically one rarely sees a change in the motor behavior of the stomach in the absence of free acid. Emptying time of the barium meal is within normal range, there is no actual dumping, and the 4–6 hour residue is about that of the normal schedule.

There are however, cases of true achylia gastrica with persistent diarrhea and loss of weight, in fact with intractable diarrhea which persists for years in spite of very large doses

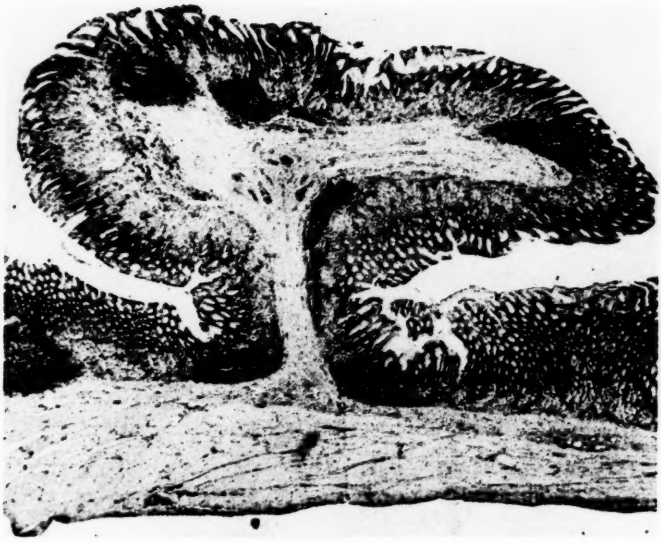


C.

Diffuse gastritis with atrophy. Mucous membrane from resected specimen of the patient whose radiograph is shown in Fig. II.

of hydrochloric acid. Fortunately, these cases are few and far between; they present very difficult therapeutic problems, for the achylia is always associated with a psychoneurotic temperament and is really only part of a much more complex generalized functional disease.

In clinical medicine anacidity or achylia gastrica has a definite connotation because of its association with the severest anemias. Complete anacidity or achylia gastrica is an essential part of the picture of pernicious anemia. Apparently there are exceptions even to what seems to be an invariable role, for Faber and Gram (12) found four cases of pernicious anemia with retained acid, and even Levine and Ladd (13),



D.

Polypoid excrescence in a case of antral gastritis simulating ulcer.

in their large series of 100 cases found some exceptions. The question always arises—are these instances of true anemia? In spite of criticism on all sides as to the authenticity of such exceptional cases, the fact remains that occasionally one meets with a case of pernicious anemia which is a true idiopathic anemia and yet has persistent secretion of hydrochloric acid. In the light of the hypothesis of Castle (14), one must say that here at least is a gastric mucosa which persists in producing free hydrochloric acid even though the anti-anemic x-factor is absent; or, if not absent, is not absorbed from the intestinal mucosa. In general, however, achylia is the most constant finding in pernicious anemia; in a large percentage of cases the achylia is dependent upon a true atrophic gas-

tritis, a gastritis which is so advanced that even in remission the acid does not return and the atrophic gastritis remains unchanged. While this is the usual and accepted viewpoint, Jones (15), of Boston, has claimed to have seen with the remission of the anemia, a return to a more normal mucous membrane, as seen through the gastroscope, and even the return of the acid secretion by the mucosa.

It would be interesting to speculate upon the relationship between the antecedent achlorhydria and the subsequent development of a pernicious form of anemia. Grave anemias occur rarely in the second or third decades of life, most often only in the later decades. And yet it has been shown that the anacidity has preceded, by years if not by the lifetime of the patient, the onset of the anemia. Authenticated instances have been cited in which the achlorhydria was clearly proven to exist at least twelve years before the onset of the anemia. It would seem as if the reserves of the body, particularly the liver, were so great that the absence of the anti-anemic factor in the gastric mucosa become operative in causing the anemia only after many years of this deficiency. Or, perhaps the anacidity, though present for many years, is not sufficiently advanced in youth but becomes effective with advancing gastritis to eventually eliminate the essential production of the true anti-anemic factor of Castle (14).

Of interest to you as actuarians would be the fact that in the families of cases of pernicious anemia a higher incidence of gastric anacidity has been found than in the controlled population. Gastric test-meals carried out on the relatives, particularly the female relatives of cases of pernicious anemia, have shown as high as 40% exhibiting anacidity. Weinberg (16) found 9 instances of achylia among 24 blood relatives of anemia patients; in one family, 3 or 4 children of the patient showed anacidity. Conner (17), at the Mayo Clinic found achylia in 25.9% of the blood relatives of persons suffering with pernicious anemia, against a control figure of 14.2%. From an actuarial standpoint, it would again be interesting to



E.

Complete atrophic gastritis in a case of pernicious anemia.

know what percentage of achlorhydria cases may eventually develop pernicious anemia. Available data does not suffice to allow of a positive answer to this query. While in charge of a large gastro-intestinal clinic, I had opportunity to watch all cases of anacidity and to obtain frequent examinations of the blood to observe whether any of the patients did develop pernicious anemia; the actual development of grave anemia was never discovered even though the period of observation extended over years. Hurst (18) observed one case of anacidity, which had been tested for twelve years, develop pernicious anemia. But no sufficiently thorough or systematic follow-up of achylia cases has as yet been carried on which will answer the question as to how many cases of achylia will eventually develop anemia.

Pernicious anemia is at times a familial disease; more than one case may be seen in a family. The disease is not hereditary but a tendency to such a constitutional defect, and certainly the anacid essential precursor, may occur as a familial incident.

As an insurable risk, the fact that an applicant was a blood relative of a patient suffering from anemia of a primary type should have much significance. Particularly so, were it known that the applicant had an anacidity.

The relationship of anacidity to sprue is an interesting one. The anemia in sprue is apparently not dependent upon the presence or the lack of hydrochloric acid in the gastric secretions. In careful studies of a series of sprue cases, no relationship has been proven to exist between the acidity and the color index in the blood. In sprue, the disturbance is one of intestinal absorption; while the anti-anemic factor may be present in sprue, the inability of the small intestine to absorb fats and vitamins and the anti-anemic x-substance elaborated by the gastric mucosa may lead to the pernicious type of anemia as well as to every manifestation of the avitaminoses. The gastric anacidity or acidity is in itself not the deciding factor as to the type of anemia.

Classical medicine associates carcinoma of the stomach with anacidity; this idea is no longer held to be true, or to be true only in part. The statement is made that 60% of all cases of gastric carcinoma are anacid, and that figure may very well be accurate. However, the remaining 40% have more or less normal acidities, a fact which, in the attempt to diagnose the presence of carcinoma of the stomach, produces much of a smoke screen. It should be reiterated and taught that advanced carcinoma of the stomach is consistent with retained acid secretion. Certainly with an Ewald test-meal the report of normal figures for gastric acidity is not unusual; if one were however to undertake a fractional test-meal, then the curve of acid in cases of carcinoma is quite characteristic. Free acid may and may not be present (it is really not very important)

but the curve of total acidity is usually high and widely separated from the line of free acid, indicating that free acid had originally been secreted but had been abundantly neutralized by some combining agency. This combining agency is a serous protein substance, possibly exuded by the ulcerating surface of the neoplasm in amounts sufficient to convert free acid into combined acid. The very fact that in most cases of carcinoma of the stomach a high total acidity curve is shown proves that acid is capable of secretion in large amounts in spite of the presence of an ulcerating new-growth. Many authorities explain the anacidity on the basis of a chronic gastritis associated with the new-growth, either as a precursor and causative agency or as a sequel to the new-growth. A. F. Hurst (18) has particularly emphasized the significance of a preceding chronic gastritis which, in his opinion, may eventually undergo a malignant cellular degeneration leading to carcinoma. He has observed verified cases of carcinoma of the stomach in which an antecedent anacidity had existed for years, apparently on the basis of a chronic gastritis. The whole concept of what constitutes gastritis, histologically and gastroscopically, is still so confused, and the relationship between anacidity and gastritis is so ill-defined that it is difficult to accept without reservations the thesis of Hurst that gastric carcinoma arises in chronic gastritis. One can readily conceive of a gastritis secondary to an ulcerating fungating neoplasm, just as one recognizes gastritis as commonly accompanying gastric or duodenal ulcer. But in these instances the gastritis is secondary to the neoplasm or ulcer and probably does not participate in any great extent in creating the condition of anacidity or acidity associated with the particular disease.

It would be practically impossible for any actuarian to attempt to establish by statement or by examination, first, the presence of a chronic gastritis with anacidity and second, to attempt to predict the probability of the origin of a gastric carcinoma on the basis of such a hypothetical inflammatory

base. Nor for that matter could anyone predict the percentage of gastric ulcers which might undergo malignant degeneration, a fact which is in the midst of debate, still lacking a conclusive answer. But at least an ulcer is something tangible that one can follow for years, while in gastritis one has an intangible, if not an untouchable point.

The antrum of the stomach is sometimes the seat of a disease in which anacidity is quite consistent, in which the radiographic picture is one of carcinoma with an antral defect, in which the symptoms are loss of appetite, pain, and often hemorrhage, much as in carcinoma. To differentiate this antral disease from true carcinoma constitutes a difficult clinical problem. The disease is not truly a gastritis because the microscopic findings vary from a hypertrophic to an atrophic gastritis; at times the mucous membrane, as visualized through the gastroscope, shows marked inflammatory changes. On the other hand, occasional polyps are thrown up which bleed; the condition has even been known to go on to partial stenosis of the pylorus. The true differentiation between antral spasm or antral gastritis and carcinoma can only be made by the gastoscopist; with a clear view of the antrum and the pylorus any doubts as to the true nature of the lesion should be abolished. The condition is frequently accompanied by an anacidity. This fact, together with a radiographic picture of a defect, led to frequent resections before the introduction of the gastroscope as a means of differential diagnosis. It is best to consider this group of cases as of benign nature and based upon a chronic gastritis associated with an anacidity.

The relation of acidity to ulcer and the importance of anacidity following partial resection of the stomach for ulcer is today a very important and a very interesting study. Ulcer is constantly associated with normal and high acidities. The object of the operation of subtotal resection is not to remove the ulcer, a procedure which in itself does not suffice to prevent recurrences, but is to accomplish a true state of gastric

anacidity. The operation of subtotal resection, when performed for *gastric* ulcer, is uniformly followed by an achlorhydria; such an anacidity is complete and under these circumstances the patient can be guaranteed against the recurrence of a marginal jejunal ulcer. Partial gastric resection for *duodenal* ulcer, in women, is also regularly followed by an anacidity. In men, however, in those who undergo resection for duodenal ulcer, about 33 1/3% retain free acid as a post-operative manifestation; it is in this group that recurrences are noted, approximating 4 to 6%. What should interest the actuarian is the presence or absence of free hydrochloric acid after the operation of partial resection for peptic ulcer. If any acid survives, the possibility of recurrence of ulcer remains open; if an anacidity is achieved, as it should be in a properly executed and adequate resection, recurrence of ulcer is out of the question; adequate digestion and good function is promised and the case can be considered from the standpoint of ulcer as a perfect future risk.

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PRESIDENT CRAGIN—Dr. Crohn, we are deeply indebted to you for a clinical paper by an insurance-minded master of this subject. It is something that we don't get too often.

We now come on to a new subject, and when one thinks of statistical studies, the name of Dr. Dublin is always brought up in the Association.

Dr. Dublin is well known to us all and his statistics as published in the Metropolitan Life Insurance Company's bulletin and various other places are always looked on as those coming from a master. This year he has come to us with an entirely new study. It is with a great deal of pleasure that I present Dr. Louis I. Dublin, Ph. D., Third Vice President of the Metropolitan Life Insurance Company, who will speak to you on "The Inheritance of Longevity—A Study Based Upon Life Insurance Records". This is done with the assistance of his colleague, Mr. Herbert H. Marks. Dr. Dublin!

THE INHERITANCE OF LONGEVITY—A STUDY BASED UPON LIFE INSURANCE RECORDS

By Louis I. Dublin, Ph. D., *Third Vice-President and Statistician* and Herbert H. Marks

Metropolitan Life Insurance Company, New York

The inheritance of longevity is a subject of wide general interest, but it is of practical importance to the life insurance business. It is not surprising, therefore, that, inside and outside the insurance companies, there have been many attempts at statistical analysis of data on longevity of parents and their offspring. There are, however, many inherent difficulties in the analysis of the problem, for longevity is not a simple unit character, but a complex of many and more or less independent elements. Adequate analysis of the problem requires the facts of longevity for at least two completed generations of a large number of families, and this involves long continued observation on many persons over long periods of time. The effects of environmental conditions on children in broken families also enter the picture. The long-term upward trend in human longevity in the last two generations is another confusing factor in such studies. In this paper, we shall not attempt to make an exhaustive or critical review of the literature but shall merely cite some of the more typical studies and their results, and shall report on a recent investigation made on records of the Metropolitan.

Early research in the field followed the relatively direct and simple lines of the collection and analysis of genealogical records. There is, for example, the study of the Hyde family by Alexander Graham Bell[1] which covered nearly 3,000 persons. Dr. Bell classified the parents and their children into groups according to their ages at death. He found that when one considered the age at death of either or of both parents, more of the children of long-lived parents survived to advanced ages than did children of short-lived parents, and in consonance with this finding, the average age at death of children of long-lived

parents was higher than that of short-lived parents.

Pearson and Beeton[2], using English genealogical records, measured the correlation between the length of life of parents and their children and also between pairs of children of the same parents. They obtained positive coefficients of correlation in both types of analyses. But it is significant that the coefficients between collaterals were higher than those between parents and children. The actual coefficients were not large and the significance of the results is rather doubtful also because of the type of material used and the method of analysis.

Pearl[3] at Johns Hopkins made many studies of the subject, one of which is based on a random sample of about 25 percent of the Family History Records of the Department of Biology at Johns Hopkins University, consisting of some 100,000 persons, resident in or near Baltimore. He computed life tables for various groups of grandparents, parents and their offspring, classified by age at death. He found that the expectations of life of parents and grandparents of children dying at ages 50 and over were higher than those of the corresponding ancestors of children dying under 50. These differences in the life table values of parents and grandparents were most marked at the older ages. Curiously enough the expectations of life for grandparents were generally greater than for parents, in both groups. Similar analysis of parents of two groups of children, widely divergent with respect to age at death, namely, those dying under five years of age and those attaining age 80, gave even more striking differences, especially at the older ages of the parents. At age 20, the expectation of life of the parents of the long-lived children was about 25 percent greater than for parents of children dying under age five, and at ages 60 and 80 these expectations were 70 percent greater. Furthermore, comparing the expectations of life of sons of three groups of fathers—(a) attaining age 80; (b) dying between ages 50 and 79; and (c) dying under 50, Pearl found a progressive increase in expectation of life with increasing age of the father at death. These differences, varying in degree, were observed at every age.

In a more recent investigation, Professor Pearl and his daughter[4] obtained the ages at death of the parents and grandparents of a group of 365 persons whose survival to age 90 or over had been authenticated. They compared the average ages at death of these forebears of the long-lived group with those for a group, consisting of 136 persons in the Family History Records whose forebears were also all deceased. The very method of selecting this second group resulted in a serious bias because a large proportion of the parents must have died at relatively early ages. There is no wonder, then, that the average and median ages of the forebears of the long-lived group were distinctly higher than those in the second group. What is of most significance is the high average ages at death of the forebears of the long-lived group, which, for each of the four grandparents, paternal and maternal, was slightly in excess of 75 years and which was 72.4 years for fathers and 73.3 years for mothers. The median ages exceeded these means by approximately four years in each case. Consistently, the averages are higher for grandparents than for parents.

Over a period of at least 60 years, the records of life insurance companies have been intermittently analyzed to discover quantitative relationships between the longevity of parents and their offspring. These records would seem to afford ideal material for such study. The application forms for insurance used by most companies have for decades contained inquiries regarding the longevity and causes of death of parents and brothers and sisters and even of grandparents of the applicant. Insurance records make available large numbers of cases containing such information and in which the subsequent mortality of one of the offspring (the insured) is known. Furthermore, the population from which insured persons is drawn is fairly homogeneous as to social and economic level.

Life insurance records, however, have certain serious defects and it will be well to consider these before summarizing

the results of earlier studies, and before presenting the findings of the present investigation of records of the Metropolitan Life Insurance Company.

Such defects in life insurance material fall under a number of heads:

1. The attitude of life insurance companies has generally been critical of applicants with a poor record of family longevity, and, consequently, many of the worst risks were either rejected or limited to substandard insurance. Since most of the studies on longevity have been made on persons accepted for standard insurance, cases of the type described would be largely eliminated from observation.

2. There is an appreciable amount of misstatement, intentional or unintentional, of ages and of causes of death of parents and of brothers and sisters. This is probably most pronounced among those with a poor family history, because the attitude of the insurance companies is known to many applicants. Family pride may also be involved.

3. Medical selection of lives for insurance introduces a definite bias, and this is probably most pronounced with persons presenting signs or symptoms of disease related in any way to the poor longevity in their family.

4. At the usual insurance ages, one or both parents of the applicant are often still living and the proportion of such parents is a function partly of his age and his order of birth. In such cases, there is no way of knowing the age at death of the parents.

5. The order of birth, which is not recorded in the aplication may itself be an important factor in the results, even though practically all applicants in these studies are adults when they enter observation.

The first important large scale investigation of the subject in this country was the experience of 34 life insurance companies (Specialized Mortality Investigation[5]) on men of average weight with short-lived parents (both dead under 60), and on those with long-lived parents (both attained age 75).

This experience covered those insured between 1869 and 1899, traced to the policy anniversary in 1900. The number of individuals in these two classes combined was about 300,000. The mortality in the group with long-lived parents was distinctly better than of the other group. We shall later comment on the results of this Investigation.

Approximately ten years later, Dr. Brandreth Symonds[6] of the Mutual Life Insurance Company made a rather elaborate analysis of the subject based on 116,590 men insured in his company between 1891 and 1899 and traced to 1909. Dr. Symonds took into consideration the age of grandparents when either living parent was under 70. If the mortality of those with an average family history be taken as 100 percent, applicants both whose parents lived to be 70 had a mortality of 95 percent of the average; those with one parent and two grandparents living to 70, the other parent living, had a mortality 93.7 percent of the average, and those with both parents living and under 70, but with three grandparents living to 70, 94.2 percent. Those with poor family histories, however, where both parents had died under 60, had a mortality 120.4 percent of the average. The mortality of those with the most favorable family history was, therefore, 25 percent better than of those with an unfavorable one.

Our first investigation of the subject was made in 1930. We re-analyzed the material of the Specialized Mortality Investigation, computing, from the printed report, the experience by attained ages. We then investigated the experience of the Company on three broad classes of insured (1) both parents living and over 50, (2) one or both parents dead at ages 50 or over and (3) one or both parents dead under 50. We based this study on white male lives between the ages of 20 and 49 at issue, insured in 1899 to 1902 and traced to the anniversary in 1928. The number of cases was approximately 70,000. We computed the death rates by attained age in the groups described and constructed life tables for them.

Our re-analysis of the Specialized Mortality Investigation showed a consistently lower death rate by attained age among

those whose parents both had reached age 75 than among those both whose parents had died under age 60. On the average, the death rate in the group with long-lived parents was 20 percent better than in the other group. The expectation of life at age 27 among the applicants with long-lived parents exceeded by about two and one-third years that of the applicants with inferior parental longevity. Even at age 47 the advantage was still as much as two years.

Among our own risks the most favorable mortality was found in the group of insured, both of whose parents were living and 50 or more years old. The mortality in the other two groups, one or both parents dead under 50 and one or both dead at 50 or over, showed no consistent differences. The expectation of life at age 22 for the insured with both parents living and past age 50 was about two and one-third years greater than for those with one or both parents dead under 50. The expectations of life among the latter group and among those with one or both parents deceased over 50 were of pretty much the same order, varying at most ages by less than .2 years.

Additional data from insurance sources were reported in 1932 by Valentine Howell[7] of the Prudential and by Charles A. Taylor[8] of the Life Insurance Company of Virginia and R. R. Benjamin[9] of the Metropolitan, who discussed Howell's paper before the Actuarial Society of America. These studies dealt with those insured at later ages and took into account the longevity of brothers and sisters as well as parents. These three reporters presented results that did not entirely accord with each other nor with those of previous studies of the subject, either because of the selected character of the material of these new studies, the limited number of lives, or the method of analysis employed. The longevity of the insured and of members of their families showed no consistent relationship.

This paper by Howell stimulated two independent studies, one by Marshall[10] of the Provident Mutual and another by Arthur Hunter[11] of the New York Life. Marshall's study is

based on the experience on issues in his company from 1908 to 1912. The first part of his study related to the mortality of the brothers and sisters of the insured at ages 20 and over, classified according to whether both parents were living, both dead or whether the father or the mother were living at the time of the issuance of the insurance, and according to the attained age of the parents. The mortality of both brothers and sisters was highest where both parents were dead, lowest where both were living and intermediate where only one was living. The mortality ratios where only one parent survived were about the same whether the father or mother was living. The mortality ratios of these brothers and sisters of the insured varied from 87 percent of the expected in the most favorable group to 114 percent in the least favorable group. The mortality among the insured lives themselves, who were traced to 1928, showed up better among those both of whose parents were living than among those both of whose parents were dead. But in the latter group, approximately the same mortality was found in the group where both parents had died under 60 as among the rest. Where both parents were living, the mortality was higher among those with parents still under 60. The experience on those risks both of whose parents had attained 70, was somewhat above 100 percent, and there was no significant difference between the groups whether either or both parents survived or both were dead.

In his study of the New York Life material, Arthur Hunter used a rating scheme in which he distinguished good, average and poor family histories. His study covered standard male risks insured during 1914 to 1917 and carried to the policy anniversary in 1931. His study showed rather small variation in the ratios of actual to expected mortality in the three classes. The mortality ratios were slightly better among those with good family histories than among those with poor family histories at all ages of issue up to 50. Beyond that age, the poor family longevity group showed the best mortality. Among those with average family histories, many of

which must be indeterminate, the mortality was generally pretty close to that of the poor family histories.

THE PRESENT INVESTIGATION

The present investigation is based upon the mortality experience among 118,000 white men in the Ordinary Department of the Metropolitan Life Insurance Company, ages 20 to 64 years at issue of insurance, between the years 1899 and 1905 and traced to the policy anniversary in 1939. Fortunately this period is one during which the mortality in middle life and old age has been relatively stable, but even the declining trends of mortality at the younger ages do not disturb comparisons because the standard used in the study is based on the contemporaneous mortality experience on all male risks. For this study, we discarded all cases—and this affected younger risks particularly—in which both parents were living and either or both were still comparatively young (sum of ages less than 110 years). We likewise discarded those cases one of whose parents was living, with the sum of parental ages less than 110 years. We also excluded cases both whose parents had died with the sum of ages at death 110 or less, if one parent had survived to age 70.

Our original plan of investigation took account of eight separate classes of parental longevity. The results for these detailed groups, however, indicated very clearly that the fundamental differences in longevity of the cases studied were dependent upon the survivorship of the parents, regardless of their age at the time of their sons' acceptance for insurance. We shall, therefore, present first our material for three broad classes of applicants, namely, those with (1) both parents living, (2) one parent living, (3) both parents dead. These three main groups were further classified for ages at issue, 20 to 29, 30 to 39, 40 to 49 and 50 to 64. And within the specified age groups, the material was considered by duration of insurance, 1 to 40 years, 6 to 20 years and 21 to 40 years.

The mortality of each group was measured against a mortality standard based on the total experience on all white male

lives with policies issued in 1899 to 1905, carried to the policy anniversary in 1939. This mortality table was graduated with slight modifications by the method evolved by Wells[12]. The results are expressed in ratios of actual to expected mortality in each group as calculated on this standard table.

At all ages combined, the lowest mortality ratio—88.4 percent of the expected—was recorded for those insured men both of whose parents were living when the insurance was issued. The highest ratio was recorded among those with both parents dead at the time the insurance was issued. The ratio in this group, 110.8 percent, is approximately 25 percent higher than in the best group. Intermediate between these, with respect to mortality, are those cases in which one parent survived at the time the insurance was issued. This group gave a slightly better than average result, namely, 97.7 percent of the expected. (See Table 1)

Somewhat more marked differences are found when the age of the insured at time of issue is considered. Among those insured between the ages of 20 to 29, the mortality in the best group (both parents living) was somewhat better than average, namely, 94.0 percent of the expected, whereas in the worst group (both parents deceased) the ratio was 121.2 percent of the expected. Again the experience in cases with only one parent surviving was intermediate between the two, but somewhat higher than average, the ratio being 106.9 percent of the expected. Thus, while the range of the ratios is not greatly different among these younger risks than at all ages combined, the ratios are all on a somewhat higher level.

With increasing age at issue, the ratios in all three groups decline, but the margins between the best and worst groups increase. Among those with both parents deceased, the figures at 30 to 39 are 115.6 percent, at 40 to 49, 111.8 percent, and at 50 to 64, 104.1 percent. Among those with both parents living, the ratios in these age groups are respectively, 88.8 percent, 78.5 percent, and 73.3 percent. The ratios in the group with only one parent surviving are in each of these age groups

TABLE 1

Mortality Among Insured Men Classified According to Survivorship of Parents When Insurance Was Issued.

Ratios of Actual to Expected Deaths Based on Contemporaneous Mortality Experience on All Male Lives.

Issues 1899 to 1905 Traced to Policy Anniversary in 1939.

Metropolitan Life Insurance Company, Ordinary Department

Duration; Age group at issue	Percent Actual To Expected Deaths			Actual Deaths		
	Both parents living	One parent living	Both parents dead	Both parents living	One parent living	Both parents dead
All Durations						
20–64	88.4	97.7	110.8	5,163	7,503	11,882
20–29	94.0	106.9	121.2	1,620	1,048	703
30–39	88.8	101.9	115.6	2,704	3,468	3,020
40–49	78.5	91.9	111.8	763	2,470	4,652
50–64	73.3	84.6	104.1	76	517	3,507
Durations 6–20						
20–64	86.2	96.7	111.1	2,165	3,303	5,766
20–29	96.5	109.2	117.4	692	441	276
30–39	86.7	104.1	119.4	1,116	1,482	1,269
40–49	69.0	88.2	113.0	314	1,107	2,173
50–64	80.7	82.1	104.0	43	273	2,048
Durations 21–40						
20–64	90.0	98.8	109.2	2,323	3,303	4,568
20–29	92.9	107.4	120.0	674	450	301
30–39	88.9	100.1	111.1	1,252	1,597	1,399
40–49	90.3	95.8	108.6	370	1,090	1,936
50–64	71.9	86.4	104.8	27	166	932

intermediate, and in the same order of ages given above are, respectively, 101.9 percent, 91.9 percent, and 84.6 percent.

Offhand, it may seem to be an anomalous situation that the mortality ratios should decline consistently with advancing ages at issue in every group. The explanation of this is really quite simple. The mortality table by which all groups are measured is, at most ages, simply the sum of the groups that we have studied, and the proportionate contribution of each group to the total varies with age. To the extent that a group represents the most typical situation with respect to survivorship of parents in a particular age span, it would contribute most of the data on mortality in that age group and, consequently, its mortality would be expected to be close to the average. Thus, for risks under 30 the typical situation is that both parents are still living, but for risks over 50 it is most likely that both have died. Consequently, on the basis of the ratios of actual to expected mortality, the advantage of the group with both parents surviving is expressed in an increasing margin with advancing age between it and the general mortality of the insured, which for each age division equals 100 percent. There may be additional significance, however, in the relatively small excess mortality among those insured at ages 50 to 64, both whose parents were deceased. The fact of survivorship in good health to age 50 and beyond is of itself an indication of good physique, quite apart from the medical screening of applicants at these older ages.

In considering the differences with respect to duration of insurance, we have, for obvious reasons, discarded the first five insurance years in which the effect of medical selection is greatest. Comparison of the broad groups of policy years 6th to 20th and 21st and over shows no important difference from the general pattern for all durations combined. Indeed, the differences, according to duration, are generally so small that we may presume that the effect of parental longevity persists and is not greatly different at older ages than it is at the younger ages.

Causes of Death

The mortality from the principal causes of death among these applicants was also considered, and these results likewise are measured against the mortality from the specific causes by age at issue and by duration among all male risks included in the standard table. These results in the cause of death analysis are also expressed in terms of the ratio of actual to expected deaths from the specific cause.

For the most part, the mortality from each of the principal causes of death of which analysis has been made, follows the general pattern for the total mortality. There are differences in degree but that is all. Surprisingly enough, this statement applies not merely to deaths from infections, such as tuberculosis and pneumonia, and degenerative diseases of the cardio-vascular-renal group, but even to deaths from suicide and accidents. Without going into details which are given in Table 2, our findings indicate that the general level of mortality, age considered, is higher among those with inferior parental history, and that while the expectation of eventually dying from degenerative diseases is higher among groups with better longevity than those with inferior longevity, the age specific death rates from such diseases are lower in these better groups than in the others.

The only other details to which we may draw attention at this time are those regarding tuberculosis and pneumonia. The mortality ratios for these causes among the younger risks (ages 20 to 29) both whose parents had died, were appreciably above those for all causes. Thus, the ratio of actual to expected deaths from tuberculosis was 138.1 percent, and from pneumonia, 130.7 percent, as compared with the general average in this group of 121.2 percent.

Experience in Detailed Groups

Let us now consider the facts in the detailed groups in which the fundamental distinction is the sum of the ages of the parents, whether living or dead. We have used the at-

TABLE 2

Comparative Mortality from Principal Causes of Death Among Insured Men. Classified According to Survivorship of Parents When Insurance Was Issued.

Ratios of Actual to Expected Deaths Based on Contemporaneous Mortality Experience of All Male Lives.

Issues 1899 to 1905 Traced to Policy Anniversary in 1939.

Metropolitan Life Insurance Company, Ordinary Department.

Age group at issue; Cause of death	Percent Actual To Expected Deaths			Actual Deaths		
	Both parents living	One parent living	Both parents dead	Both parents living	One parent living	Both parents dead
Ages 20–64						
All Causes	88.4	97.7	110.8	5,163	7,503	11,882
Tuberculosis (all forms)	82.3	100.8	118.5	544	666	783
Cancer (all forms)	91.8	103.1	106.8	511	818	1,199
Cardiovascular-renal diseases	86.5	97.1	109.6	1,745	2,906	5,060
Diabetes	93.9	99.6	102.2	109	154	204
Pneumonia (all forms)	88.5	90.2	112.7	413	551	991
Suicide	88.9	96.0	118.8	129	161	229
Accidents	93.4	99.6	110.8	351	431	587
Ages 20–29						
All Causes	94.0	106.9	121.2	1,620	1,048	703
Tuberculosis (all forms)	89.5	106.6	138.1	272	182	139
Cancer (all forms)	92.5	112.5	134.6	112	79	56
Cardiovascular-renal diseases	95.1	109.8	111.0	431	287	172
Diabetes	86.7	106.2	122.8	23	16	11
Pneumonia (all forms)	89.1	101.4	130.7	128	83	63
Suicide	88.3	97.6	148.3	46	29	26
Accidents	108.2	105.9	114.7	147	81	52
Ages 30–39						
All Causes	88.8	101.9	115.6	2,704	3,468	3,020
Tuberculosis (all forms)	77.3	103.8	116.5	228	328	273
Cancer (all forms)	90.2	106.7	116.7	286	385	328
Cardiovascular-renal diseases	87.2	102.2	115.0	944	1,260	1,108
Diabetes	104.2	105.8	93.5	69	78	53
Pneumonia (all forms)	97.2	93.9	116.6	229	247	234
Suicide	92.1	95.3	139.5	67	76	84
Accidents	91.9	102.8	112.7	176	216	179
Ages 40–49						
All Causes	78.5	91.9	111.8	763	2,470	4,652
Tuberculosis (all forms)	69.4	91.9	117.2	41	144	274
Cancer (all forms)	98.7	101.6	100.1	107	307	472
Cardiovascular-renal diseases	76.7	92.3	111.2	332	1,117	2,104
Diabetes	73.3	89.3	107.7	16	51	91
Pneumonia (all forms)	59.3	83.2	118.4	46	175	378
Suicide	68.6	98.6	106.1	13	49	78
Accidents	63.9	93.5	111.8	28	112	205
Ages 50–64						
All Causes	73.3	84.6	104.1	76	517	3,507
Tuberculosis (all forms)	*	68.3	105.5	3	12	97
Cancer (all forms)	60.1	78.3	104.5	6	47	343
Cardiovascular-renal diseases	76.0	83.7	104.3	38	242	1,676
Diabetes	*	103.8	99.1	1	9	49
Pneumonia (all forms)	103.4	82.4	101.5	10	46	316
Suicide	*	81.8	98.6	3	7	41
Accidents	—	84.2	105.9	—	22	151

* 5 deaths or less.

tained age at time of issue of the sons' insurance for living parents and the age at death in the case of deceased parents. For the three broad groups—both parents living, one living, and both dead, we have the following divisions of parental longevity:

Sum of ages 110 to 124
Sum of ages 125 to 139
Sum of ages 140 or over, sub-divided into:
(a) Both attained 70
(b) Others in this class
(c) Both attained 75

For the group with both parents dead, we have also these divisions:

Both died under 45
Both died under 55 or the sum of ages under 110
(a) Excluding cases where one parent attained 60
(b) Excluding cases where one parent attained 70

Finally, we have the combined experience for all those cases where the sum of the parents' ages, whether living or dead, was 140 or over. For most groups with either or both parents dead, we also tabulated the material excluding cases in which either parent had died by violence or the mother died from causes associated with childbearing. The details are omitted from this paper, because these exclusions had no material effect on the results.

The ratios in these detailed groups, summarized in Table 3, bring out clearly our earlier statement that the fundamental distinction in the mortality is associated with the survivorship of the parents, either or both. For, the mortality ratios tend to be alike, regardless of the attained ages of parents, within each broad group—both parents living, one living and both dead. This is true generally for each subdivision by age of the insured and duration of insurance. The exceptions are relatively few. Some of these may be significant. Others are very questionable because of the small size of the samples which make the probable errors of the mortality ratios in

TABLE 3

Mortality Among Insured Men Classified According to Survivorship and Longevity Record of Parents When Insurance Was Issued.

Ratios of Actual to Expected Deaths Based on Contemporaneous Mortality Experience on All Male Lives.

Issues 1899–1905 Traced to Policy Anniversary in 1939.

Metropolitan Life Insurance Company, Ordinary Department.

Parental survivorship status; Sum of parental ages **	PERCENT ACTUAL TO EXPECTED DEATHS														
	Durations 1–40					Durations 6–20					Durations 21–40				
	Age Groups at Issue					Age Groups at Issue					Age Groups at Issue				
	20–64	20–29	30–39	40–49	50–64	20–64	20–29	30–39	40–49	50–64	20–64	20–29	30–39	40–49	50–64
Both Parents Dead															
Total	110.8	121.2	115.6	111.8	104.1	111.1	117.4	119.4	113.0	104.0	109.2	120.0	111.1	108.6	104.8
Under age 45	111.1	105.7	107.1	119.6	109.6	118.3	114.1	119.4	125.3	107.0	101.6	96.2	95.0	112.4	107.3
Under age 55, or sum of ages less than 110,															
a) except if one parent attained age 60	113.4	121.5	110.3	115.1	108.7	119.5	124.3	119.9	127.0	102.7	104.8	116.6	99.7	104.0	110.9
b) except if one parent attained age 70	111.1	121.9	109.8	112.9	102.7	115.8	125.1	117.3	123.0	98.1	104.7	112.4	102.3	104.6	104.9
Sum of ages 110–124	112.1	113.9	119.9	110.6	106.3	110.5	88.7	123.1	110.2	105.1	112.7	126.5	116.7	109.9	108.0
Sum of ages 125–139	111.6	120.7	120.1	109.3	107.8	110.7	120.4	111.9	110.5	109.7	113.1	138.8	125.3	105.5	110.8
Both attained age 70 or more	109.7	182.2	120.6	115.9	102.2	106.0	*	128.9	109.2	100.5	111.9	*	113.1	118.5	103.0
Sum of ages 140 or more, except if both attained 70	107.2	170.7	112.7	111.8	99.4	111.4	187.5	132.1	110.9	105.4	101.4	*	92.0	109.4	93.1
Both attained age 75 or more	112.9	*	142.5	128.1	99.7	105.4	–	139.1	116.4	96.8	122.0	*	134.0	140.7	101.7

TABLE 3———(Continued)

Parental survivorship status; Sum of parental ages **	PERCENT ACTUAL TO EXPECTED DEATHS														
	Durations 1–40					Durations 6–20					Durations 21–40				
	Age Groups at Issue					Age Groups at Issue					Age Groups at Issue				
	20–64	20–29	30–39	40–49	50–64	20–64	20–29	30–39	40–49	50–64	20–64	20–29	30–39	40–49	50–64
One Parent Dead, One Parent Living															
Total	97.7	106.9	101.9	91.9	84.6	96.7	109.2	104.1	88.2	82.1	98.8	107.4	100.1	95.8	86.4
Sum of ages 110–124	99.8	109.9	99.1	95.0	75.8	99.5	106.5	101.9	93.5	70.0	99.4	117.2	94.3	98.0	75.7
Sum of ages 125–139	99.6	97.8	105.5	95.6	80.0	102.4	114.2	109.4	95.9	75.5	97.7	82.8	104.2	95.5	78.5
Both attained age 70 or more	90.4	110.7	100.1	85.5	85.5	86.5	115.5	97.3	81.8	81.3	95.8	84.8	105.0	90.5	92.3
Sum of ages 140 or more, except if both attained 70	95.7	126.2	103.2	88.2	94.6	87.8	109.8	101.9	72.9	97.8	104.1	160.8	105.2	100.7	94.8
Both attained age 75 or more	86.5	*	109.0	80.5	78.1	84.3	*	111.7	78.4	74.1	88.8	*	114.8	77.9	82.6
Both Parents Living															
Total	88.4	94.0	88.8	78.5	73.3	86.2	96.5	86.7	69.0	80.7	90.0	92.9	88.9	90.3	71.9
Sum of ages 110–124	89.1	91.6	87.9	71.3	—	88.5	91.9	86.9	58.8	—	90.7	94.3	88.3	91.3	—
Sum of ages 125–139	87.7	96.1	88.4	75.0	*	87.1	104.7	87.0	68.1	*	86.5	85.0	87.7	84.0	*
Both attained age 70 or more	87.9	125.6	91.3	82.6	78.4	83.9	144.4	87.0	75.5	83.1	93.6	97.8	95.2	93.4	83.0
Sum of ages 140 or more, except if both attained 70	88.8	103.3	92.1	79.5	*	74.1	85.2	83.6	56.8	*	95.6	126.3	88.0	97.8	*
Both attained age 75 or more	84.3	209.2	83.2	86.8	70.6	73.8	*	82.3	67.5	72.2	99.6	*	87.6	112.9	74.3
Both Parents Attained Age 70 or More, or Sum of Ages 140 or More, Whether Living or Dead	98.9	120.7	101.2	97.1	97.4	96.0	116.6	100.9	90.2	98.5	101.3	123.6	100.4	102.7	96.4

TABLE 3———(Continued)

Parental survivorship status; Sum of parental ages **	ACTUAL DEATHS														
	Durations 1–40					Durations 6–20					Durations 21–40				
	Age Groups at Issue					Age Groups at Issue					Age Groups at Issue				
	20–64	20–29	30–39	40–49	50–64	20–64	20–29	30–39	40–49	50–64	20–64	20–29	30–39	40–49	50–64
Both Parents Dead															
Total	11882	703	3020	4652	3507	5766	276	1269	2173	2048	4568	301	1399	1936	932
Under age 45	449	82	158	154	55	206	35	67	72	32	183	33	72	64	14
Under age 55, or sum of ages less than 110,															
a) except if one parent attained age 60	2258	387	834	718	319	1032	159	358	345	170	923	161	374	297	91
b) except if one parent attained age 70	3143	434	1117	1080	512	1453	179	472	527	275	1287	174	517	445	151
Sum of ages 110–124	2760	156	802	1063	739	1284	49	340	480	415	1124	76	370	464	214
Sum of ages 125–139	2877	87	695	1211	884	1396	36	266	561	533	1132	43	347	509	233
Both attained age 70 or more	1897	9	225	772	891	996	3	101	362	530	617	3	98	302	214
Sum of ages 140 or more, except if both attained age 70 or more	1205	17	181	526	481	637	9	90	243	295	408	5	67	216	120
Both attained age 75 or more	928	2	88	366	472	488	–	37	173	278	293	1	37	142	113

TABLE 3———(Continued)

Parental survivorship status; Sum of parental ages **	ACTUAL DEATHS														
	Durations 1–40					Durations 6–20					Durations 21–40				
	Age Groups at Issue					Age Groups at Issue					Age Groups at Issue				
	20–64	20–29	30–39	40–49	50–64	20–64	20–29	30–39	40–49	50–64	20–64	20–29	30–39	40–49	50–64
One Parent Dead, One Parent Living															
Total	7503	1048	3468	2470	517	3303	441	1482	1107	273	3303	450	1597	1090	166
Sum of ages 110–124	2826	680	1456	620	70	1195	270	616	276	33	1275	312	659	279	25
Sum of ages 125–139	2667	294	1323	929	121	1210	140	576	432	62	1155	108	613	396	38
Both attained age 70 or more	1166	25	394	545	202	531	12	161	255	103	502	7	194	231	70
Sum of ages 140 or more, except if both attained 70	844	49	295	376	124	367	19	129	144	75	371	23	131	184	33
Both attained age 75 or more	476	5	135	218	118	222	3	58	104	57	198	2	67	84	45
Both Parents Living															
Total	5163	1620	2704	763	76	2165	692	1116	314	43	2323	674	1252	370	27
Sum of ages 110–124	2252	1057	1154	41	–	928	442	471	15	–	1031	459	548	24	–
Sum of ages 125–139	1761	451	1021	285	4	755	203	427	122	3	770	169	467	133	1
Both attained age 70 or more	792	53	345	327	67	349	26	145	141	37	355	17	159	154	25
Sum of ages 140 or more, except if both attained 70	358	59	184	110	5	133	21	73	36	3	167	29	78	59	1
Both attained age 75 or more	287	10	92	140	45	120	3	40	53	24	137	5	43	72	17
Both Parents Attained Age 70 or More, or Sum of Ages 140 or More, Whether Living or Dead	6262	212	1624	2656	1770	3013	90	699	1181	1043	2420	84	727	1146	463

* Five deaths or less.
** At death or at time of son's application for insurance.

such groups rather large in relation to the differences between them and other risks.

The first exception to the general pattern which we believe to be significant is the mortality ratios for the group with one parent dead, but both attained 70 or 75. The ratios are appreciably better than for the rest of the class with one parent dead, and approximate the results for those with both parents living at these ages. Fundamentally these groups are quite alike.

Another consistent departure from the general picture is that younger risks with aged parents show high ratios of actual to expected deaths. This is most pronounced at ages 20 to 29 at issue, but in some instances is apparent at ages 30 to 39 also. This characteristic has been noted in earlier investigations also, and in our judgment, it indicates a generally inferior vitality in such children. The causes that bring about a high maternal mortality among older mothers and for their infants probably affect the vitality of the children who survive, although the economic and other advantages of longer established families would seem to act favorably on such children. There may be another explanation, namely, that a great many of these people are the later born children of large families, and since such families are more frequent in the lower socio-economic groups, the higher mortality found may be the reflection of poor standards of living in spite of the good record of parental longevity. This factor may also influence the mortality in certain other groups where the ratios are not in line with what we would expect.

The converse of this situation—exceptional vitality of children born to young parents who survived to the maturity of their children—cannot be traced directly in our material. There may be some indication of it in the very low ratios of mortality among those insured at ages 40 to 49 years the sum of whose parents' ages, both still living, was from 110 to 139 years and also for those insured at ages from 50 to 64, whose parents were both living at age 75 or over. Among the groups

for which we have fair-sized samples, these had the lowest mortality.

What was uppermost in our minds and will be in the minds of most of those who study these findings is the comparatively small gradation, or its absence, in the detailed parental longevity groups within the three broad parental survivorship classes. So far as the groups with both parents dead are concerned, the factors which we have mentioned above may be in part responsible, but it is in these groups that the frequency of misstatement is greatest. We have no idea as to its extent because seldom is any attempt made to authenticate the assertions of the applicant regarding the ages at death of his parents.

Within the other groups, the prime factor is that we are dealing for the most part with family histories of indeterminate quality and that, to a great extent, these groups overlap. Thus, when consideration is given to the probabilities of survivorship, individual and joint, it will be realized that most of the individuals whose parents are both living and the sum of whose ages is 110 to 124 will eventually graduate into groups with parents both living, the sum of whose ages will reach 125 to 139, and the majority of the latter will graduate into the class with parents both surviving with the sum of their ages 140 or over. The corresponding situation holds for the half-orphan groups.

Causes of Death

The mortality from the principal causes of death in the detailed classes tends, just as in the broad parental survivorship groups, to follow the trend of the total mortality. This affords additional confirmation of our impression that the groups with superior longevity, are resistant both to infection and to early onset of degenerative disease. By and large, with the relatively small numbers of deaths in some classes when broken down by cause, the deviations from the general mortality ratio are seldom significant. The contrasts again in the detailed parental longevity groups were usually greater with respect to

TABLE 4

Comparative Mortality from Principal Causes of Death Among Insured Men.
Classified According to Survivorship and Longevity Record of Parents When Insurance Was Issued.
Ratios of Actual to Expected Deaths Based on Contemporaneous Mortality Experience on All Male Lives.
Issues 1899–1905 Traced to Policy Anniversary in 1939.
Metropolitan Life Insurance Company, Ordinary Department.

Parental survivorship status; Sum of parental ages **	PERCENT ACTUAL TO EXPECTED DEATHS							
	Causes of Death							
	All causes	Tuber-culosis	Cancer	Cardio-vascular-renal diseases	Diabetes	Pneumonia	Suicide	Accidents
Both Parents Dead								
Total	110.8	118.5	106.8	109.6	102.2	112.7	118.8	110.8
Under age 45	111.1	152.8	126.1	102.4	87.5	112.1	69.0	105.2
Under age 55, or sum of ages less than 110, except if one parent attained 70	111.1	131.5	111.8	104.6	102.9	112.3	121.1	119.0
Sum of ages 110–124	112.1	119.0	107.2	115.2	137.3	118.0	112.1	99.0
Sum of ages 125–139	111.6	114.5	103.4	113.1	100.1	107.7	105.3	97.1
Both attained age 70 or more	109.7	101.9	100.0	110.9	81.2	105.0	121.4	120.3
Sum of ages 140 or more, except if both attained 70	107.2	100.4	111.9	99.1	54.8	125.7	156.6	132.2
Both attained age 75 or more	112.9	110.3	106.1	111.9	87.2	104.5	125.7	147.7
One Parent Dead, One Parent Living								
Total	97.7	100.8	103.1	97.1	99.6	90.2	96.0	99.6
Sum of ages 110–124	99.8	109.2	107.0	100.1	90.7	93.7	99.9	96.0
Sum of ages 125–139	99.6	95.3	104.6	96.8	112.4	96.0	96.8	113.4
Both attained age 70 or more	90.4	87.7	86.9	91.2	100.2	78.4	94.8	82.8
Sum of ages 140 or more, except if both attained 70	95.7	99.1	111.0	98.4	87.5	78.6	80.2	91.9
Both attained age 75 or more	86.5	104.1	86.2	71.9	*	83.8	124.7	95.9

TABLE 4———(Continued)

Parental survivorship status; Sum of parental ages **	PERCENT ACTUAL TO EXPECTED DEATHS							
	Causes of Death							
	All causes	Tuber-culosis	Cancer	Cardio-vascular-renal diseases	Diabetes	Pneumonia	Suicide	Accidents
Both Parents Living								
Total	88.4	82.3	91.8	86.5	93.9	88.5	88.9	93.4
Sum of ages 110–124	89.1	81.7	92.4	92.3	95.0	82.2	75.2	87.8
Sum of ages 125–139	87.7	85.0	89.9	81.4	90.0	98.6	95.0	97.4
Both attained age 70 or more	87.9	83.4	88.0	85.5	88.5	88.7	96.9	101.7
Sum of ages 140 or more, except if both attained 70	88.8	70.8	106.9	82.5	118.1	77.7	139.5	97.7
Both attained age 75 or more	84.3	67.4	79.9	90.3	*	76.4	108.0	69.9
Both Parents Attained Age 70 or More, or Sum of Ages 140 or More, Whether Living or Dead	98.9	91.9	99.6	98.0	85.4	95.9	112.7	105.9
	ACTUAL DEATHS							
Both Parents Dead								
Total	11882	783	1199	5060	204	991	229	587
Under age 45	449	54	52	162	7	36	6	24
Under age 55, or sum of ages less than 110, except if one parent attained 70	3143	290	328	1201	57	254	70	182
Sum of ages 110–124	2760	187	277	1214	64	237	51	122
Sum of ages 125–139	2877	168	282	1280	48	227	47	120
Both attained age 70 or more	1897	81	180	865	24	155	32	94
Sum of ages 140 or more, except if both attained 70	1205	57	132	500	11	118	29	69
Both attained age 75 or more	928	39	90	416	12	74	15	54

TABLE 4———(Continued)

Parental survivorship status; Sum of parental ages **	ACTUAL DEATHS							
	Causes of Death							
	All causes	Tuber-culosis	Cancer	Cardio-vascular-renal diseases	Diabetes	Pneumonia	Suicide	Accidents
One Parent Dead, One Parent Living								
Total	7503	666	818	2906	154	551	161	431
Sum of ages 110–124	2826	314	301	1030	52	210	67	166
Sum of ages 125–139	2667	218	292	1013	62	204	57	170
Both attained age 70 or more	1166	72	121	502	25	81	23	53
Sum of ages 140 or more, except if both attained 70	844	62	104	361	15	56	14	42
Both attained age 75 or more	476	32	51	175	5	37	12	25
Both Parents Living								
Total	5163	544	511	1745	109	413	129	351
Sum of ages 110–124	2252	276	206	731	46	166	51	156
Sum of ages 125–139	1761	184	177	576	37	158	47	123
Both attained age 70 or more	792	57	84	313	16	64	18	49
Sum of ages 140 or more, except if both attained 70	358	27	44	125	10	25	13	23
Both attained age 75 or more	287	15	29	130	4	21	7	12
Both Parents Attained Age 70 or More, or Sum of Ages 140 or More, Whether Living or Dead	6262	356	665	2665	101	499	129	330

* Five or less deaths.

** At death or at time of son's application for insurance.

acute infections than to cardiovascular and other degenerative diseases and to deaths from violence, but even from these causes the mortality ratios were lower in the groups with low ratios from all causes. The details of this part of our analysis are presented in Table 4 for all ages at issue combined.

Mortality in Relation to Causes of Death in Family of the Insured

In the course of the investigation, the insured men reporting deaths in the family from certain causes—cardiovascular-renal diseases, cancer, diabetes and suicide—were picked out for study. In analyzing this material, we did not use the classifications by age at death of the parents, as was done in the main part of the study. Moreover, cases with brothers or sisters of the insured dying from these special causes were included, and thus, in the special analyses, we have cases which were discarded from the main study. Except in the group with a family history of cardiovascular-renal diseases, the special analyses were limited to cases with only one relative dying from the specified disease, because cases with more than one such relative were comparatively few, and we preferred to have groups that were homogeneous in this respect. As, in the main body of the study, these analyses are limited to those insured between ages 20 and 64.

Cardiovascular-renal Diseases

This aspect of the study was limited to those in which there were two or more deaths at ages under 60 years among parents or brothers and sisters of the insured. Despite the large number of cases in our main investigation, and despite the broad definition of this disease group, there were only 325 cases from the entire experience that qualified for this analysis. In only eleven cases were there three relatives, within the limits defined, deceased before age 60 and none with more than three. These cases would be rare because such a history would be likely to cause rejection for standard insurance. In slightly less than half of the total cases, both parents had died from one of these conditions. Most of the

remaining cases were equally divided with respect to a family record of these diseases among (a) father and one sibling; (b) mother and one sibling; and (c) two siblings.

Of this group of insured, 89 have died, giving a ratio of approximately 140 percent of the expected number. The mortality ratio was somewhat better for durations beyond 20 years than for durations shorter than this. When ages at issue are considered, the ratios showed a definite increase with advancing age. Deaths from cardiovascular-renal diseases accounted for 46 of the 89 deaths, as against only 26 expected. The ratio of actual to expected deaths from these causes, approximately 175 percent, is appreciably higher than for all causes combined and, indeed, accounts for the greater part of the excess mortality of the group.

It should be noted that the mortality ratios, both for all causes combined and for cardiovascular-renal diseases, are higher for this group than for those insured persons in the main body of the study who reported both parents dead at the time of application for insurance.

Cancer

This aspect of our study is based upon 4,174 cases with one death from cancer in a parent, brother or sister. It would have been desirable to make detailed studies according to the site of the cancer reported in the deceased relative, but the number of cases where this fact was given was insufficient except with regard to cancer of the stomach.

The analysis of the cancer material undifferentiated with respect to site was made separately for three groups according to the relative affected, namely, father, mother, and brother or sister. It was found that, although there was no marked difference in the mortality from all causes of death for these groups there was a decided difference in the levels of cancer mortality. Among those risks whose mothers had died of cancer, there were 58 deaths from cancer among the insured, or 133 percent of the expected. These ratios were not significantly different for those insured under 40 and those insured

over that age. The cancer mortality of those whose fathers had died of cancer was very slightly above normal, 108 percent, based on 32 deaths. Again, there was little difference according to the broad age groups at issue under and over 40, but the number of cancer deaths in the former group was quite small. Those insured who reported a brother or sister as having died from cancer, had a cancer mortality somewhat below the expected—84 percent, based on only 16 deaths from this cause.

Among those applicants with a family history of cancer of the stomach, both the mortality from all causes and the mortality from cancer, all forms, was practically normal. But of the total deaths from cancer, 26 in number, the stomach was the site in 14 cases, and this number was approximately one-third above the expected. While this result is suggestive, the number of deaths is too small to be statistically significant.

Diabetes

The experience on the 873 insured with a single death from diabetes in the family was analyzed with respect to the relative affected, namely, father, mother, and brother or sister, and for all these groups combined, according to the build group of the insured. It was found that those insured whose mother had died from diabetes had a mortality ratio significantly above average, namely, 125 percent, based upon 55 deaths. The ratios were quite similar at ages under and over 40 at issue. The mortality in the other two groups with a diabetic relative was approximately average, namely, 104 percent, based on 73 deaths among those with the father dying from diabetes and 98 percent, based on 54 deaths, among those with a brother or sister dying of the disease. The differences in the ratios by broad ages at issue were not significant.

There were only six deaths from diabetes altogether and, consequently, the differences between the various sub-groups are unimportant. While these six deaths were 179 percent of

the expected, this result cannot be deemed entirely significant.

When these risks were classified according to their build, the mortality ratios were found to increase moderately with increasing weight, but, on the whole, the differences were not more marked than one would expect among those with no death from diabetes in a close relative. Thus, the mortality ratio for underweights was 97 percent of the expected, for average weights, 108 percent, and for overweights, 114 percent. The differences in the ratios between underweights and overweights was somewhat smaller among those under age 40 at issue than those past that age. Curiously enough, of the 6 deaths from diabetes, 4 of them occurred among those underweight at issue of insurance.

Because of the frequency of premature arteriosclerosis in diabetics, the mortality from cardiovascular-renal diseases was analyzed in these various groups. In general, the same characteristics as for all causes combined, but somewhat accentuated in degree were noted for this disease complex. Among those with diabetic mothers, the mortality from cardiovascular-renal diseases was quite high—168 percent of the expected—based on 26 deaths, but among those with diabetic fathers, the excess was not significantly large and among those with a diabetic brother or sister, the ratio was actually below average—76 percent of the expected—based, however, on only 16 deaths. When the cases were classified by build, irrespective of the relative affected, overweights were found to have a 40 percent excess mortality from cardiovascular-renal diseases, whereas among the average weight men, the mortality was normal—99 percent—and better than average among underweights, 88 percent. These differences again are probably not much larger than would be found for those giving a family history negative with respect to diabetes.

Suicide

The experience on 837 men with a family history of suicide was studied separately for two groups—(a) those reporting a parent and (b) a brother or sister dying in this manner.

Attention was paid in this analysis to the mortality from accidents as well as that from all causes and from suicide. These risks had a mortality from all causes very slightly above normal, namely, 103 percent of the expected for those with a history of suicide by a parent and 107 percent in the sibling group. No differences in mortality in the parent group were found between the two broad age groups at issue, under 40 and 40 or over, but in the sibling group, the mortality among those under 40 was rather high, namely, 130 percent, based on 41 deaths. The older risks, in this group, however, had a mortality slightly better than normal—93 percent. The mortality from suicide was relatively high, although the actual number of such deaths was not large. In the total experience combined, 14 of the 92 deaths were due to suicide. This number was nearly four times the expected. This ratio was approximately the same among those insured under 40 as among those insured past that age. The ratio was higher, but probably not significantly so among those with a brother or sister dying from suicide than in the parent group. The mortality from accidents was, at all ages combined, found to be a little better than average, 82 percent, based, however, on only eight deaths. This was due to the very favorable experience among those at ages under 40 at issue. Among those insured beyond that age, however, were recorded six of these accidental deaths, giving a mortality ratio above normal, but not significantly so in view of the small number of deaths involved.

Conclusions

This analysis points broadly to a positive relationship between the longevity of parents and offspring. For the purposes of life insurance underwriting, our findings indicate that credit can be given for a favorable family history only when both parents are living, or, at the older ages at issue, if one parent is living and is at an advanced age. This credit for a favorable family history is not applicable, however, to younger risks whose parents, whether living or dead, are reported to

have attained advanced ages. For various reasons these cases give unfavorable mortality. There is, likewise, no call for credit for a favorable parental longevity if both parents are reported as deceased at high ages unless there is definite proof of these ages and the applicant falls outside the group of children born to relatively old parents.

On the other hand, there is warrant for a small rating on most applicants under age 50 if both parents are dead. A somewhat higher rating is indicated for such applicants under age 30 than for those between 30 and 50. Our results indicate that not much weight should be given to the reported ages of the parents at death unless the facts are substantiated.

With respect to the ratings for particular diseases in the family history, we have not much to contribute, for several reasons. Within the limited field of our work, a few interesting and significant items appeared. They indicate that the conditions studied by us do not generally affect our ratings, because the mortality from all causes in these groups showed little deviation from the expected, although often the mortality of the insured was high from the same cause that was responsible for the death of a parent or sibling. The reason that this did not raise the mortality perceptibly was that the individual disease caused a relatively small fraction of the total mortality. The only significant exception to this was for the cardiovascular-renal group, which had been most severely selected. The results for that group indicate that with two or more immediate relatives dying prematurely from such disorders, a moderate rating is warranted. While the insured whose mother died from diabetes showed a moderately high mortality, we do not recommend any rating until this fact is confirmed by further investigation.

From a more general point of view, we pose the question whether such association as exists between parental longevity and that of offspring is, in the main, the result of inheritance rather than of environmental conditions. Our data afford little or no evidence on this matter, nor do the basic

records lend themselves particularly to such analysis. There can be no question, however, of the distinctly bad effects of orphanhood on the health and mortality of young people, and these effects may be quite lasting. The premature death of either parent often results in the breaking up of the home, with its consequent lack of care of offspring. This usually applies whether it is the father or mother who dies. The situation is even worse when children are left complete orphans at a relatively early age. The children in all broken families where either or both parents have died, are, on the whole, much more poorly housed, badly fed and poorly clothed than other children, and more of them are compelled to go to work at an early age; consequently more engage in unskilled labor and other poorly paid work than the children in normal families. All of these conditions were worse at the beginning of the century than they are today.

On the other hand, there may be strong beneficial influences from survivorship of parents on the longevity of offspring even at comparatively late ages of the offspring, and these influences may have nothing whatever to do with heredity. For example, parents in these cases may help their offspring financially and in other ways, and the good mental and spiritual environment of a family, particularly if it is closely linked together, in which the parents survive to late ages is real and not without effect on the mental and physical health of their children.

Another general aspect of this subject, to which we may draw attention is the relatively limited gain in longevity likely to accrue from control of hereditary factors as compared with the large additions achievable by control of environment. Even if all the advantage in the groups with the lowest mortality in our study were the effect of heredity alone, the maximum difference in expectation of life at age 20 probably does not exceed three years. This figure is relatively much smaller than the gain of five years which has been recorded among men in the general population at age 20 in the short period covered

by the present study. This clearly indicates that recent gains through improvement in environment greatly exceed those which have accrued from favorable heredity. And the possibilities of gain in life expectation through changes in environment have by no means been exhausted. Intensive application of our present medical and sanitary knowledge would result in such a large decline in death rates that six years more would be added to the current expectation of life at age 20. These facts have a distinct bearing on the controversy that has gone on for many years between the leaders of the eugenics movement, on the one hand, who stress the importance of heredity and, on the other hand, physicians, social workers and other groups who have emphasized the social gains inherent in the improvement of the environment. Certainly, on the basis of conditions in the world today, the environmentalists have distinctly the better of the argument in so far as this relates to the question of longevity.

While, therefore, we have demonstrated certain variations in mortality according to parental longevity, we have perhaps been a bit ambitious in labelling our paper "The Inheritance of Longevity." The study of our own results and those of other investigators of life insurance material make it doubtful whether insurance material alone can throw much light on the subject of the inheritance of longevity. In fact, such studies in man may all be quite inconclusive, partly because of the effects of environment which cannot be disentangled or evaluated with reasonable accuracy and also because there is little conscious selective mating, in humans, of individuals of long-lived stocks. Indeed, marked deviations from average ancestral longevity must be relatively infrequent if one goes back more than two generations. As Pearson pointed out long ago, the farther back one goes, the greater is the likelihood that the numerous ancestors of any individual represent an average sample of the population with respect to longevity. Exceptions to the rule exist, but they are, in our judgment, so comparatively infrequent as to be of little consequence in the selection of applicants for life insurance.

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PRESIDENT CRAGIN—Once in a while the maker of a program runs into a piece of extraordinary good fortune. In seeking a discusser of Dr. Dublin's paper, we have been fortunate to find a man who is a definite factor in the United States Public Health Service and the National Institute of Health; for fourteen months in charge of investigations in geratology at the National Institute of Health; at present organizing and developing an entirely new unit to be concerned with the problems of the aging in the broadest aspect; since July, part-time consultant of geratology, interested particularly in what one might call personal preventive geriatrics; at present also editing a new textbook on geriatric medicine, which should appear on the book shelves next spring.

It is a great pleasure to present to you to discuss Dr. Dublin's paper, Dr. Edward J. Stieglitz of Washington, D. C. and Chicago.

DR. STIEGLITZ—Dr. Dublin's splendid paper has covered the subject of hereditary factors in human longevity so comprehensively and so well that there is little left for a discussant to say. From the strictly actuarial viewpoint these data are significant chiefly in that some additional ratings appear justified for younger applicants if both parents are dead, whereas the cases in which credit might be warranted are far more limited. We all know the frequency of vague and indefinite information as to the age and causes of death of parents and concerning the past history, as elicited by the examining physician. These data are, as Dr. Dublin points out, definitely weighted in life insurance examinations and, therefore, require substantiation before credit in premium rating is feasible or logical. However, similar errors will be found in any series of examinations of people, whether they be insurance, pre-employment or voluntary clinical examination. I do not feel that the selective weighting is of any great magnitude. We all forget quickly that which we do not want to remember. The major source of error is ignorance and wishful thinking, rather than deliberate misstatement. The usual clinical history obtained from the usual patient is full of inaccuracies. For

many years, in teaching senior medical students, I have tried to bring this point home by a very simple demonstration. Upon asking these young people, almost all in their twenties, how many of them can state the causes of death of all four of their grandparents, or of as many who have not survived to date, less than 10 percent of the class are able to answer affirmatively. Yet these men have been unusually conscious of the importance of heredity in their own lives and have been taught to be observing and to search for facts. I used to point out to them the necessity of great skepticism in interpreting hereditary influences, for, after all, even if the information was forthcoming and could be assumed to be accurate (which is very doubtful), the data involved but two generations and from the genetic viewpoint this is far too small a sample to be significant. Humanity is such a heterogenous mixture that genetic analysis has immense probabilities of error in anything but large series, such as this presented today.

I am particularly pleased that Dr. Dublin expressed doubt as to whether the association which does exist between parental longevity and that of offspring is the result of biologic inheritance or a matter of environment. The battle between the eugenecists and environmentalists still rages though the ultimate outcome is quite clear: neither can win for both are partly right. As both factors are present and as it is impossible to separate them, except in a very few isolated and exceptional cases (so few that the observations will always be interpreted to fit in whichever thesis the observer is trying to defend), why is it not simpler to include both types of influences under one title of "familial influences"? From the pragmatic viewpoint of a clinician attempting to clarify the predisposing etiologic background of any given disorder and from the viewpoint of actuarial analysis for life insurance medicine, such inclusion of both sets of factors under a single head avoids much misunderstanding and confusion. Can we definitely prove that obesity is entirely an inherited characteristic in the purely biologic sense of inheritance? We cannot, for the overweight offspring is reared in an environment of overeating

and overindulgence and thus acquires habits in infancy and youth which persist throughout life. Similarly, familial vulnerability to the development of hypertensive arterial disease may be partly a question of inheritance in "the quality of the rubber," as Osler put it, but it may also stem from habits of apprehension, worry, hyperconscientiousness and dietary and other indiscretions acquired in youth. Dr. Willius has stated that coronary disease is more frequent among smokers; perhaps smokers are more frequent in those whose constitutional and emotional characteristics predispose to coronary arteriosclerosis. When it is so difficult to determine which comes first, which is the horse and which is the cart, it seems wiser to consider them both as a "horseless carriage" in which vehicle and motive power are combined. The individual, after all, is not a static unity. Each and every experience, reaction, infection, intoxication, psychic and physical trauma or fatigue, which constitute the countless vicissitudes of existence, alter and modify the inherent and intrinsic capacities to change the individual.

The tremendous and significant gains in average longevity and life expectancy in the last forty or fifty years are certainly not due to any radical departure from former methods of selection in human mating. The quaint old custom of falling in love has not altered appreciably, nor is it likely to. Dr. Dublin stated (I quote): "And the possibilities for gain in life expectation through changes in environment have by no means been exhausted." Therein lies the most significant message in Dr. Dublin's paper. It is a justified challenge to preventive medicine. Though there is no question that more intensive application of existing knowledge and methods of public health, sanitation, industrial hygiene and the like, can but further improve the situation, THESE WILL NOT BE ENOUGH. It is notable that although life expectancy at birth increased some 14 years in the quarter century from 1910 to 1935 (from 46.3 to 60.2 or 30 percent), life expectancy at 50 years increased but 2.8 years (from 18.3 to 21.1 or 15 percent). There is reason to pause and consider why

the gains have been so asymetric.

(1) There are two methods of approach to preventive medicine:—the impersonal, "wholesale" or public health type and personal or "retail" attack. Impersonal public health activities have included institution and control of modern sanitation, enactment and enforcement of quarantine regulations, control of avoidable hazards of atmospheric contamination in industry, schools, public buildings and homes and the application of mass immunization. These methods have assisted personal preventive medicine in causing magnificent improvement in the health of youth, but alone they could not have accomplished what has come about. Of equal significance has been the energetic program of personal prophylactic medicine carried out by pediatricians in taking well babies and making them healthier by proper feeding, personal parental instruction in hygiene and careful periodic inventory with immediate correction of minor defects. The impersonal and indirect forms of preventive medicine have not proven to be productive of much benefit to the health of mature adults. They are effective only against exogenous disorders, infective or environmental in etiology. They are applicable only to relatively homogenous groups, where a minimum of individualization is necessary. The common diseases of those past the meridian, such as cardiovascular–renal disease, diabetes mellitus, the arthropathies, cancer and gout, are not exogenous. They arise from within. Many are etiologically associated with indulgences. With aging comes increasing individual variation. These diseases are insidious of onset, slowly progressive and usually cause long periods of disability before they finally destroy. They are not florid, acute, communicable, self-limited and immunizing as are those of youth. The early detection of the occult functional defects which herald the beginnings of degenerative disease is difficult, requiring time and the highest type of diagnostic acument. Thus personal preventive medicine for adults is inevitably more expensive than the wholesale approach. Nevertheless, it is the ONLY FEASIBLE AP-

PROACH TO PREVENTIVE GERIATRICS. The periodic health inventory and health maintenance consulation is the keystone to the arch of preventive medicine for adults to bridge the dangerous years from 40 to 60, and thoroughness and individualization are the twin foundations.

The potentialities of preventive geriatrics are as yet largely undeveloped. (2) Exploration of this field is becoming increasingly urgent with the increasing millions of elderly people in our population, for disablement from the chronic and progressive disorders of these years is a grave economic menace. (3) The health of a nation cannot be measured by mortality tables alone.

Increased longevity and life expectancy is not only a challenge to clinical preventive medicine but also to the leaders in insurance medicine, educators and all those responsible for the guidance of the people in matters of health. With every privilege arise obligations. The immeasurably valuable privilege of increased longevity carries with it the personal obligation of effort toward health maintenance and the general obligation of organized health agencies to make available facilities to assist such efforts. Longevity without health is more than a personal and individual tragedy; in the aggregate it becomes a dangerous economic burden upon the Nation. We can ill afford additional loads now. Defense, like therapeutics, should be more than heroic attack against exogenous menaces. Defense must consider the internal milieu of the body politic as the physician is concerned with the homeostatic mechanisms of the sick organism. Not infrequently the most precarious period of an illness is after the crisis.

Thus it behooves insurance medicine to aid in the development of the potentialities of personal preventive medicine for adults, through its investigative, educational, industrial (4), and service programs. The objective of prophylactic geriatrics is not merely the prolongation of life but the maintenance of health, vigor and productive usefulness until senility normally becomes infirmity. It must not be forgotten that the

policyholders of life insurance are aging and vulnerable to the disorders of later life. Statistical science has gone far in actuarial research to guide in the selection of risks. The maintenance of the health of these risks after they are insured is a fertile field ready for planting. The harvest will be fine in proportion to the intensity and intelligence of cultivation. Lastly, let me remind you that the wisdom, experience and judgment of older men in the positions of gravest responsibility represent an invaluable national resource. Conservation of the functional capacities of these difficult replaceable individuals by prophylactic geriatrics is a national service.

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PRESIDENT CRAGIN—I wish to thank Dr. Stieglitz very much indeed for his fine presentation, and we appreciate very much the time that he has given to us to come here and talk with us on a subject which I don't think has ever been spoken about before.

We are getting a little short on time, so I would like to ask Dr. Dublin if he has anything to say in closing.

Dr. Dublin, I want to thank you and Mr. Marks for bringing before us this most original and interesting paper. We certainly appreciate it very much indeed.

Our next topic will be a new one for this Association. We have been very fortunate in getting our speaker to come be-

fore us and bring us a little advance information on what is going on in the Statistical Bureau of the Census in Washington at the present time, and what the plans are for the future.

Our speaker has had a varied education, holds a number of degrees, including a Ph. D. from the University of Minnesota, has had a large experience in teaching medicine, vital statistics, medical statistics, and since 1935 has been chief of the Division of Vital Statistics, Bureau of the Census, Department of Commerce, Washington, D. C. He is a member of numerous societies and organizations, published a large number of articles in the field of anatomy and medicine, hospital statistics, vital statistics, and so forth.

Dr. Halbert L. Dunn will now address us on "Vital Statistics and Life Insurance in a Changing World." Dr. Dunn!

VITAL STATISTICS AND LIFE INSURANCE IN A CHANGING WORLD

By Halbert L. Dunn, M. D.
Chief of Division of Vital Statistics
Bureau of the Census, Department of Commerce,
Washington, D. C.

My subject is Vital Statistics and Life Insurance in a Changing World.

I suppose no one here would care to deny the fact that it *is* a changing world. However, most of us would have different ideas as to the degree of change that is now in progress. Many of us probably believe that after the present disturbances are over, everything will be just the same. Personally, I doubt it. Some of the forces which are at work at the present time are tremendous. They will affect life insurance quite as much as the lives of all of us as citizens.

Of course, the thing which we see most clearly is the war itself. At the present moment we, too, are on the brink of such a war with all its implications. If we lose in that war, life insurance could well be wiped out as such. In a totalitarian state there is not much place for life insurance, because insurance and security become a part of the state itself. There are other profound forces at work in addition to the war. Some of them are involved with the limitations in our national resources which are needed as a basis of national security, some with the factor that Dr. Dublin has just discussed, namely, the aging of our population. As he said, we can be sure that the people of the United States will be considerably older thirty years from now, that the numbers and percentages of people over sixty years of age will be markedly greater.

This age factor is a serious threat to the life insurance business. You will undoubtedly see the crude death rate

go up instead of down. What does that mean to dividends? What does it mean in terms of the shifting characteristics of the causes of death which we face? Longevity brings with it a larger exposure to the killing diseases of old age.

Another great force which is in operation now is the urge of people all over this country, in fact, all over the world, for personal security. That force is a momentous one. Our social security movement springs from it. Pension plans, the Townsend movement, security against war, and, in fact, the entire insurance business, are part of it. The urge to security is primal in character. What does that mean with reference to insurance?

Perhaps the underlying factor of change behind all these various movements is science itself. It is science which makes possible the destructive forces now unleashed in war; which releases control of power to individuals and to nations in a degree which has hitherto been unknown; which makes possible, by increased production, the survival of increasing numbers of people upon this earth of ours; and which has shrunk the distances of the earth by improved transportation and communication facilities.

These forces turned loose by science will bring even more change in the future. Production and possibilities for destruction will be multiplied. New social controls must be devised or mankind will destroy itself in this rapidly changing world that we are in. Life insurance also must strike a new balance and achieve new concepts if it expects to survive.

The subject of vital statistics is going through a profound change at the present time. Before we consider all of the factors and relationships involving both vital statistics and life insurance, I would like to sketch for you briefly the picture as I see it for the vital statistics system of the country.

For those who are not familiar with the system, I will give a brief description. It is in essence a federal-state system. The offices of the state registrars of vital statistics are

located in the state health departments with but one exception – Massachusetts – which places this office under the control of the State Department. Four cities independently handle vital statistics separately from the states: Boston, New York, Baltimore and New Orleans. The District of Columbia is treated separately as a state.

The relationship between the Division of Vital Statistics of the Bureau of the Census and the state bureaus of vital statistics is on a co-operative basis. Transcripts of birth, death, marriage and divorce are bought by the Census Bureau from the states. It is the States' responsibility to collect, process, and make accurate the certificates. That procedure is accomplished by paying local agents a fixed price for the certificates which they obtain. The family doctor is the most important person in filling out the birth and death certificates. It is his responsibility to fill out the entire birth certificate and the medical part of the death certificate. It is the funeral director's responsibility to make out the personal particular portion of the death certificate; he cannot bury the body until he has produced a death certificate.

This is a brief description of the system as it stands at present. It has many weaknesses; yet it is a system to which many of us look for valuable information and upon which all of us as citizens depend for essential documents for proofs of facts concerning birth, marriage, divorce and death. Jobs, security and privileges often depend on the proof of such facts.

Many organizations are interested in the data collected by this vital statistics system. Inside the federal government, and in addition to the Bureau of the Census, the agencies most interested are the United States Public Health Service, the Children's Bureau, and the Social Security Board with its bureaus of Employment Security and Old Age and Survivor's Insurance, the latter needing proof of the facts of death before

payments are rendered. The Army and Navy have become especially interested in the proof of the facts of birth, particularly in as much as they indicate United States citizenship.

Many people do not have birth certificates on file. Somewhat less than half the people of the country (approximately sixty million) do not possess birth certificates. This is due to the fact that birth registration was not officially launched for the country as a whole until 1915. For those people who do not have birth certificates on file, national standards have recently been developed. Health agencies have agreed to follow the minimum requirements laid down in these standards for filing delayed birth certificates. Sufficient documentary evidence must be submitted by the citizen before a delayed certificate is placed on file.

After these recommendations for the filing of delayed certificates were approved by interested organizations, the Army and Navy became interested in them as a defense measure. Over the combined signatures of the Secretaries of War and Navy there has been mailed, to every contractor and subcontractor dealing with defense orders, a recommendation that they use these standards.

There are other agencies inside the government which depend upon this vital statistics system; the Budget Bureau, the Executive Office of the President, the Council for National Defense; the United States Civil Service Commission, the Work Projects Administration, the Department of State, the Department of Justice, the Railroad Retirement Board, the Veterans' Bureau, the Office of Education, and the Department of Agriculture. The United States Civil Service Commission requires a properly filed birth certificate for every person going into federal service. The WPA has spent millions of dollars in the last five or six years trying to strengthen the vital statistics system by building important indices for birth, death and marriage certificates. The Department of State requires that the death certificate and facts of death of any citizen of a foreign country who has died in the United States must be transmitted to the country

of which he was a citizen. The Department of Agriculture is involved with population problems in the rural district.

At the state levels, the interested organizations include the state and county health offices, the state and city registration bureaus, and those portions of the federal agencies mentioned above which have their sub-offices in the states.

Among the non-official organizations of a national character, the American Public Health Association and the American Statistical Association are particularly interested in population statistics. Also, the American Bar Association has finished its consideration of a uniform vital statistics law. The American Medical Association has always been concerned with vital statistics because of the large part played by the physicians of the country.

As I stated previously, it seems to me and to many of us that the federal-state system is a weak organization to do the problem that it has to do. The offices are not well staffed, and the volume of record work is tremendous. Bureaus of vital statistics are breaking down under the strain. Queue lines of people have formed in places in an effort to obtain certified copies of birth certificates or to file delayed certificates of birth.

What are some of the defects of the present system? First of all, inaccuracies. Dr. Dublin emphasized this in his talk. These inaccuracies are involved particularly with the physician's cause of death statement, and to a certain extent with the statements of the personal particulars on the birth and death certificates. Many a person as he gets a certified copy of his birth certificate has been surprised at the errors which he finds listed thereon. This happened to me about two years ago, when upon application for a certified copy of my own birth certificate I found out that I was a year and a quarter younger than I thought I was, and that my name wasn't spelled in the same way in which I had been using it for over forty years.

There are a number of reasons why the cause of death statements are wrong. First of all, the doctor is frequently

careless. In addition to that, he knows that the certificates pass under the eye of a local registrar who might well be a curious person and a gossip. Isn't it the doctor's responsibility to protect his patient's family from this annoyance? Then, in addition, the doctor does not want his patron's family to suffer because he has returned a cause of death which might militate against their securing the insurance or pension benefits of the deceased.

I know a colored physician in one of our big cities who has never put down "tuberculosis" as a cause of death, but has always called it pneumonia. Is the doctor justified in this attitude? Have insurance policies been so written by some companies as to leave loopholes which will militate against the family of the deceased in settlement? I don't know, but in any case fear of this is one of the factors which must be faced if the problem of inaccuracies in the cause of death statement is to be solved.

The second major point is non-uniformity; non-uniformity of both records and methods. Non-uniformity is particularly serious when one desires to use the birth certificate filed in one state as proof of the facts of birth needed in order to secure employment in another state. Sometimes a person can observe every rule of proper filing within his own state and still find that a certified copy of such a certificate will not be honored because of the differences between states in their practices and procedures.

The next defect is incompleteness in registration of the facts of birth and death. In view of the public pressure focused upon the necessity for birth certificates at this time, you would think that the birth of every new baby in the country would now be registered. However, in a nationwide test in 1940, just tabulated, it is evident that approximately a quarter of a million youngsters are not having their birth certificates placed on file even yet. They are going to experience the same or possibly even greater difficulties in the future in obtaining these documents simply because negligence, ignorance and lack of funds have made it impossible to record their births.

One reason for incompleteness in birth registration is that many states follow the practice of paying the local registrars from county funds. When it is impossible for the county to pay such sums of money, tax warrants are issued, registrars are not paid, and citizens must suffer the consequences.

Personnel inadequacies is another weakness. In state after state the vital statistics officials are subject to dismissal because of political upheavals. Within the last nine months the entire forces of three large vital statistics departments have been dismissed en masse. It took a long time to train those people. Substitutes, for the most part, do not have qualifications to do the job.

There is much duplication and overlapping of effort between local communities, states and the federal government. At each of these levels statistical personnel are attempting to make tabulations. Some sort of division in labor is needed so that wastage of funds will not result from overlapping. Even with such overlapping, there is an insufficiency of statistics available to the state health officers, such as the absence of population bases between census periods in order to make adequate birth and death rates or current statistical reports for county and city areas.

Another serious fault of the system is the fact that these valuable records themselves are not properly preserved. We think of them as snug in their steel vaults, safe from fire and flood. Actually, this is only true in certain states. Other factors causing deterioration are poor grades of paper and inks which fade.

Yet another defect is the apparent unwillingness on the part of state and local officials to enforce the registration law. Something must be done to strengthen enforcement, if these documents are to be depended upon to serve the citizens of the country.

Perhaps the most alarming symptom of decadence in the system is the recent practice of levying excessive charges for the services rendered in obtaining certified copies or for the filing of delayed certificates of birth. Some states require

court procedure. In one western state, for instance, the cost to place on file a delayed certificate of birth is approximately $75, since it is necessary to pay both a legal and court fee. Rackets are developing by leaps and bounds so that in many places the citizen is persuaded that it is practically impossible for him to obtain the essential proofs he needs unless he pays additional fees to some private individual who acts as his agent.

Rackets deserve special mention because sometimes they cater to falsification of certificates. The establishment of personal identity has never been a part of the birth certificate. An agency or business organization accepting such a birth record as proof of the facts of birth does not know whether or not the certified copy submitted belongs to the person using it. The Robinson couple offered a famous example illustrating this possibility of fraud. The Robinsons obtained a passport and went to Russia. There they were discovered to be spies of a European country. Yet they had United States passports. How did they obtain them? They had both applied for birth certificates of children who had died at a young age. The information used in their applications for certified copies had apparently been obtained from the death columns in the newspapers filed at the Public Library. In complete security they had then applied for the birth certificates of persons whom they knew were dead. Their fraud could not have been detected by the present vital statistics system.

If fraud is to be stopped, the knowledge of the fact of death must in some way be re-entered on the birth certificate. This, I believe, is one of the most important elements that should come into the future workings of the vital statistics system of the country. There must be some sort of central index of deaths which will make possible the notation of the fact of death upon the birth certificate. Other important files dealing with individuals, such as your insurance files, social security records, etc., would likewise benefit from such a national index of death information. There is no way for me to estimate the amount of money that must be

lost to all agencies dealing with annuities and benefit payments for lack of this knowledge. It must run to tens of millions of dollars each year. Its result is a levy against all United States citizens in overcharges for benefit costs.

If war comes, we will have still another set of problems facing the vital statistics system of the country. Canada offers us a preview of what will happen. Under the Canadian National Registration Act, passed in August of 1940 for men and women over sixteen years of age, the vital statistics system was asked to produce immediately monthly lists of deaths of everybody over sixteen years of age and to send such lists to the Dominion Bureau of Statistics. Next, it was called upon by the Provincial Division Registrar of the Department of National War Services to send monthly lists of deaths for all single and widowed men in the draft ages 19 to 45. It was then required that they inform all persons who married that it was necessary for them to file these facts with the Dominion Bureau of Statistics. This was essential, since a change in marital status altered obligations and payments, and since the Bureau needed to know all changes of address.

In addition, a whole new set of documentation was required by the Army, in particular that dealing with such facts as: identity of the soldier, who his dependents were, whether or not he was married, and to whom payments should be made in case of his sickness, death, or change in military status. These factors were particularly important because wholesome morale of both soldier and civilian depended upon them.

I should now like to turn from vital statistics for a few minutes so as to consider the problems of insurance in connection with this picture. Some of the basic problems facing the insurance companies arise from similar factors as those affecting vital statistics. For instance, how are we going to anticipate the effect of the aging population of the country upon the insurance business? How are we going to face, and what are we going to do about this nation-

wide urge toward security, which is a paramount thought in the minds of people and is becoming a major issue in both government and business? What do insurance companies face with respect to the vanishing income from their investments? Interest on capital and sound investment has been the rock upon which the present insurance structure has been built. What is the effect of world disorder, and what will be its outcome?

For the doctors who make up the membership of this organization, I would like to pose a question which particularly concerns them, i. e., of how much value is the medical examination required before writing the insurance contract? It seems to me, as an outsider, that the medical program of the life insurance companies has, for the most part, been directed at the selection of risks which are good, yet there are other elements involved which seem as important, if not more so, than that of selecting a good risk.

Certain medical problems of life insurance overlap with those of vital statistics. One of the most important of these involves the inaccuracies in the cause of death statement on the death certificate. I realize that the possibility of a confidential death certificate is a red flag to this group, but it is also true that you desire accurate knowledge concerning the cause of death. Perhaps you cannot have one without the other. That question must be answered by your group because it can't be answered by anybody else. The essential reason why the results from use of a confidential statement of the cause of death have not been adequately evaluated is due to the desire to avoid offense to the life insurance companies. Now you are coming to a time when accurate knowledge of the true causes of death will be of extraordinary importance to your business. Already you are worrying about the advancing mortality rates of heart and of the vascular renal group. It is in these classifications of death where deceit is most prevalent. How much of a problem is heart disease? We can't seriously face that question without trying to find out how good the data are. I don't see how this problem

can be faced unless you are willing to assist in evaluating the true facts.

Another overlapping problem in which you have a stake is the lack of uniformity of vital statistics records. Frequently you wish to collect from a variety of states certain documentary evidence involving proof of birth or death. Such documents should be consistent.

You are interested in certain of the facts produced by vital statistics. This is particularly true of data on the older ages, which are linked to your need for information concerning institutionalization. Advancing age and social change bring with them the increased use of institutions.

How much of the hypertension problem which has been discussed today is due to the worries and distractions of neuroses? This possibility should be a subject for your research. Dr. Parney, Chief of the Division of Industrial Hygiene, Department of Pensions and National Health, at Ottawa, Canada, has studied that problem and reported his findings. He showed that neuroses alone can produce an average debility greater than any disease, except perhaps tuberculosis and severe cardiac disturbance.

The stoppage of fraud, both with respect to a client's age and personal identity might well prove to be the greatest single service which vital statistics can offer to life insurance. A central national file for deaths would probably be almost as valuable as the one you now use dealing with disabilities reported on previous examinations.

There are certain other things with which vital statistics does not concern itself but which it seems to me should be a part of your program. For instance: What about the small policy which has always cost so much and about which so many questions have arisen in the minds of people? The usual small policy is almost completely absorbed by the funeral director. Is this sound? Is it in line with the national urge for personal security?

What about the medical care program? Sooner or later the country will probably develop some type of social or state

medicine. Will you have a part in it? If not, why not? Isn't it a part of the need for security to which the insurance business has dedicated itself?

What about the advantage of annual medical examinations? Why do you not champion that cause? It has been demonstrated that about two years could be added to the life expectancy if an annual physical examination and remedy of defects could be provided. That would mean a tidy sum of money for your companies, and at the same time would be considered a great public contribution. Why aren't you interested in this matter? The resultant savings would surely rival, if not exceed, those involved with medical selection of applicants for insurance.

Obviously I have raised many questions which cannot be answered. Forgive me for my impertinence. You might as well ask me "What are you going to do to improve the vital statistics system?" With reference to this, a stirring event occurred last week. The state health officers have come to a realization of the importance of strengthening the vital statistics system. They have officially asked that a committee be appointed, a presidential committee, if possible, which would examine what should be done and recommend a solution. I think that you folks should do something of this type. You, too, need to make a searching survey of your present activities and interests. I would like to see you appoint a committee—a committee that is capable of approaching some of these problems without bias, and with technical competence. The committee would need funds. It should develop a staff. It should serve the joint interests of this group of medical directors. Perhaps such a committee would eventually become an institute such as those serving the steel or oil industries. Why not? Life insurance is big business. It should have that sort of service. Such an institute could be constantly searching for the truth behind current trends. It would have tremendous prestige with business, government, and the public. It could ask those pointed questions

which so frequently neither business, government, or the public are willing to face alone.

PRESIDENT CRAGIN—We certainly have had our ideas clarified on the subject of vital statistics, always so vital to insurance. A clear plan is always welcome. Thanks for bringing us such a plan and putting such information before us, and also thank you, Dr. Dunn, for shedding your incognito and appearing before us under your true name and age.

Peripheral arterial disease has been claiming more and more attention from the profession in the last few years. We know very little about it from the insurance standpoint.

Our next speaker is a graduate of the University of Nebraska, and after his internship, became a Fellow at the Mayo Clinic under Dr. George Brown. Dr. Brown was probably the greatest stimulator of investigation of peripheral vascular disease in this country, and upon his death our speaker was appointed to succeed him as head of the Section of Peripheral Vascular Disease. He is Associate Professor at the Graduate School of Medicine of Minnesota, Past Chairman of the Section of Peripheral Arterial Disease of the American Heart Association and on the Editorial Board of the American Heart Association. At the present time, he is Secretary of the Section on Pharmacology and Therapeutics of the American Medical Association. He is also the author of many articles in the field of circulatory diseases. Dr. Edgar V. Allen!

CHRONIC OCCLUSIVE PERIPHERAL ARTERIAL DISEASES

By Edgar V. Allen, M. D.
Division of Medicine, Mayo Clinic
Rochester, Minnesota

I shall limit myself in this paper to a discussion of two common peripheral arterial conditions, thrombo–angiitis obliterans or Buerger's disease, and arteriosclerosis obliterans.

SYMPTOMS

These two conditions have common symptons, which are chiefly those of impaired arterial circulation to the peripheral parts. Impaired arterial circulation produces coldness of the skin but many people who have cold skin have normal arterial circulation.

Pain is a very important manifestation of impaired arterial circulation, and the most characteristic pain is intermittent claudication, which is the counterpart of the syndrome angina pectoris. It is entirely pathognomonic of impaired arterial circulation. There need not be any confusion of the symptom of intermittent claudication with any other condition, for it occurs only in conditions characterized by impaired arterial circulation. It is a symptom which is brought on entirely by exercise and relieved promptly by rest. It does not have any relation to cramps, which occur during the night while patients are at rest in bed, nor to the static distress of flat feet or metatarsalgia. Intermittent claudication is characteristically a pain which is brought on by walking. It affects the calves of the legs and disappears promptly on cessation of the activity that produces it. The patients do not need to sit; they do not need to wait long for relief, which occurs promptly. Intermittent claudication may occur in regions other than the calves of the leg, although the calves are the most commonly affected. It may affect

the arch of the foot, the digits, the wrist, the palm of the hand, the arm and forearm, and occasionally it may involve the thigh and as high as the waist in high occlusion of the aorta.

The pain which occurs with rest is of three types, generally. The first is a type of pain which occurs without any evidence of impaired nutrition of the skin, without any breaks in the skin, without any ulceration and without any gangrene. It is essentially the same type of pain which occurs in the presence of gangrene, for it is a manifestation of dying tissue. In the same connection is a pain which is a manifestation of ischemic neuritis, a very definite clinical syndrome. This pain affects chiefly the feet and occurs in paroxysms, chiefly nocturnal in origin, which are usually very severe and at times are extremely difficult to control.

Then there is, of course, the pain associated with gangrene, which is well localized to the gangrenous region and with which most physicians are entirely familiar. One word of caution is necessary about the pain which occurs before the presence of gangrene. Because this pain is associated with redness, it is commonly mistaken for pain due to inflammation. Hence, incisions are commonly made into the painful digits, chiefly the toes, or the toenails are removed, with the result that gangrene invariably occurs because of poor circulation. The one clue to the fact that this is not a pain due to some deep-seated infection is the fact that the digit is cold, rather than warm, as one ordinarily would expect when there is inflammation.

PHYSICAL FINDINGS

In arteriosclerosis obliterans and thrombo-angiitis obliterans the findings are several on examination; one of these is the change in color which occurs with the change in posture. When the extremities of patients who have impaired arterial circulation are elevated, there is unusual pallor. This may be diffuse or may be spotty. Spotty pallor indicates involvement of the digital arteries in a spotty manner, and if one

obtains arteriograms of these patients, one sees the evidence of arterial involvement. When the feet are in the dependent position, they assume a reddish, cyanotic hue which is also characteristic of impaired arterial circulation but may occur in some other conditions as well.

When patients are overweight or when the ulnar artery is deeply placed, it is often difficult to feel pulsations in it. Under these circumstances, a test, which was given my name by Sir Thomas Lewis, is helpful. The patient clenches his fist, then the examiner occludes the radial artery with his thumb and the patient opens his hand. When there is a normal arterial inflow through the ulnar artery, normal color comes back quickly. When the ulnar artery is occluded, the hand maintains its pallor, because the ulnar artery is occluded by disease and the radial artery is occluded by the examiner's thumb.

Students are taught in medical school and in their internships and residencies a great deal about examination of the heart. That is as it should be but they are taught almost nothing about palpation of the peripheral arteries and other methods of examining the peripheral circulation which are very important. When students come to me, I am amazed at their lack of knowledge about these subjects and yet it is relatively easy to become expert in these matters by repeated routine examination of patients. Any physician actively engaged in practice should become as adept at examination of the peripheral circulation as he is at examination of the heart. As a very minimum he should know how to palpate pulsations in peripheral arteries.

Two other findings are important in the presence of impaired arterial circulation. One of these is the delay in return of color which occurs when the extremities are moved from the elevated to the dependent position. Ordinarily normal color returns about ten or fifteen seconds after the feet or the hands are moved from the elevated to the dependent position, but in the presence of impaired arterial

circulation, return of normal color is greatly delayed, sometimes as long as a minute. Another similar test is the filling of the veins. Ordinarily when one moves the elevated extremities to the dependent position, the veins fill promptly (within ten seconds) but since the arterial circulation is greatly impaired in the two diseases under discussion, filling of the veins occurs much more slowly; sometimes, twenty, thirty, forty or fifty seconds are required.

A final finding in impaired arterial circulation is the presence of gangrene. Unfortunately, we see too much gangrene. Gangrene is almost uniformly an indication that the patient has not had adequate care.

Almost all the findings referable to the extremities in impaired arterial circulation are either visible or detectable by palpation. Instrumental methods are chiefly for the purpose of acquiring chemical and physiologic knowledge relative to impaired arterial circulation, but the experienced clinician needs only his hands and his eyes to enable him to make the diagnosis and to follow rather accurately the progress of the disease.

MEDICOLEGAL ASPECTS

A few remarks relative to the medicolegal aspects of these two diseases may be pertinent. At the Mayo Clinic we see a good many patients who come to us for an expert opinion relative to whether or not gangrenous lesions, such as are commonly seen, were acquired primarily as a result of trauma, that is, from crushing blows or freezing, or whether they occurred because there was some primary impairment of the arterial circulation. It is usually true that these patients have other evidence of impaired arterial circulation in addition to gangrene. They get these gangrenous lesions because their arterial circulation was poor when trauma was sustained. In Minnesota the winter is rather cold and yet we see very few frozen extremities. When we do see them, we are very suspicious that the affected person had impaired

arterial circulation before he was subjected to cold. The usual story is that a man adequately clothed, working with a group of associates who are also adequately clothed, acquires a lesion. Under those circumstances and if his associates did not acquire lesions such as he did, then one may reasonably assume that he had impairment of his arterial circulation first. On examination usually one finds impairment of arterial circulation in other extremities, so that the evidence is clear that the lesion is a result of an inherent disease of the arteries plus trauma and is not a result of the trauma alone.

THROMBO-ANGIITIS OBLITERANS

Thrombo-angiitis obliterans is a disease of unknown cause, which is characterized by inflammation of the arteries and veins of the extremities, as the term "thrombo-angiitis obliterans" indicates. It is not arteritis, but angiitis, indicating inflammaiton of the veins as well as of the arteries. It is probably an infectious or viral disease, although the evidence is not clear on this. One may occasionally culture streptococci from acute vascular lesions in thrombo-angiitis obliterans, but one cannot do this routinely, and it has been impossible to reproduce lesions in animals with any degree of constancy, as a result of injection of organisms which have been cultured from acute lesions. The most significant observation in this connection is that of a Scotch surgeon who acquired thrombo-angiitis obliterans of the hand as a result of having pierced the flesh of one finger by a spicule of bone while he was amputating the toe of a patient who had thrombo-angiitis obliterans.

The pathologic changes of thrombo-angiitis obliterans are well known. There is a diffuse inflammatory lesion of artery and vein. Cellular infiltration, proliferation of the intima, thrombosis and formation of new arteries are common.

The symptoms of thrombo-angiitis obliterans are those which I have given as common symptoms of both thrombo-angiitis obliterans and arteriosclerosis, with the exception that

superficial phlebitis occurs in about 40 per cent of instances of thrombo-angiitis obliterans, but, of course, does not occur in arteriosclerosis obliterans, which is solely an arterial disease. In both these diseases, sudden arterial occlusion indistinguishable from arterial embolism may occur and a rather large number of these subjects have the Raynaud phenomenon. From the standpoint of life insurance examiners, one of the most important observations is that the disease is not limited to the extremities routinely but may affect vessels in other places. In about 3 percent of instances there is cerebral thrombosis, and the commonest cause of death in thrombo-angiitis obliterans is myocardial infarction. Surprisingly, when myocardial infarction occurs, the lesion present in the coronary arteries is not characteristically that which is seen in the peripheral vessels but is usually a degenerative lesion.

Sometimes objections are made to the prolonged treatment of patients who have thrombo-angiitis obliterans, because physicians occasionally say, "The patient has to have an amputation sometime; let us amputate his legs and get it over." That is a fallacious statement, because many patients who have thrombo-angiitis obliterans, perhaps 90 percent, never need to have amputation of an extremity. Another observation that is pertinent is that amputation of the extremities does not solve the problem, for the disease may progress to involve cerebral or coronary arteries.

ARTERIOSCLEROSIS OBLITERANS

Arteriosclerosis obliterans is a different disease in many ways, but so far as its effects on the circulation are concerned, it is the same as thrombo-angiitis obliterans with the exception that phlebetis does not occur. The cause here is the cause of arteriosclerosis elsewhere, whatever that may be. Just why the lower extremities are more commonly affected with arteriosclerosis obliterans than any other part of the body, with the exception of the coronary arteries, is difficult to say, but it is probably intimately connected with the much higher arterial pressure in the lower extremities when man is in the upright

position. It is my impression that arteriosclerosis of certain types is intimately connected with a deranged metabolism of fat.

Diabetes, which occurs very commonly with arteriosclerosis obliterans is probably a contributing factor to the development of these arterial lesions.

The symptoms and physical findings of arteriosclerosis obliterans are the same as in thrombo-angiitis obliterans, except again that phlebitis does not occur. It is always important to search for evidence of diabetes in the presence of arteriosclerosis obliterans.

Two other statements may be made about arteriosclerosis obliterans: one does not use typhoid vaccine for elderly patients, or uses it very hesitatingly, and one does not use sympathectomy quite as freely or as quickly as one would in the treatment of thrombo-angiitis obliterans.

TREATMENT

One of the things which has been learned over a period of years is that patients who have impaired arterial circulation must not under any circumstances use tobacco in any form. There is a suspicion that in thrombo-angiitis obliterans the tobacco may have something actually to do with the production of lesions in the arteries, although this cannot be proved, but in both conditions, smoking greatly impairs the arterial circulation. When patients smoke cigarettes, the temperature of the skin decreases, indicating impaired circulation. These decreases of circulation last from fifteen minutes to an hour, and a man who smokes twenty or twenty-four cigarettes a day has permanent, persistent impairment of his arterial circulation regardless of whether or not he has any organic lesion in his extremities.

Prophylaxis in the presence of these diseases characterized by impaired circulation is important, and the prophylaxis is just the same type as has been so well outlined by many who have been interested chiefly in diabetes.

Diet does not have any value in the treatment of these conditions, except in arteriosclerosis obliterans, in which there

is occasionally an increase of the concentration of cholesterol and fat in the blood. Occasionally one encounters rather marked reduction of the concentration of the fats in the blood as a result of dietary restriction of fat, but the evidence is not at all clear that this reduction of concentration of fats in the blood prevents the progression of the organic arterial disease. One would like to think that the problem here is as simple as is the treatment of diabetes, that if one reduces the concentration of fats to normal, then the disease is well under control, but at the present time this evidence is lacking.

A great number of methods have been used to increase the circulation. The physiologic studies which have gone on in this direction have accumulated gradually over a period of years. There are several methods of applying heat locally to the extremities, but I should offer one warning: if the heat exceeds 95° F., the harmful effects are greater than the beneficial effects. Therefore, all units for applying heat directly to the extremities should be watched to see that the temperature does not exceed 95° F.

Contrast baths have had vogue for a period of years, but I do not have any evidence that contrast baths do anything for the patient under any condition, except to keep him aware of the fact that he has impaired arterial circulation. That in itself may be important.

Postural exercises—elevating the extremities and keeping them there for one minute and then having them in the dependent position for one minute—have also had a good deal of vogue. There is no evidence to my knowledge that this improves the peripheral arterial circulation, but many patients think that this treatment is beneficial. I think its chief function is in keeping the patient aware of the fact that arterial circulation is impaired.

There are a good many mechanical methods of treatment, of which the Sanders' bed at the present time enjoys a good deal of vogue. This is a lazy man's way of taking postural exercises, for this bed automatically puts head down and head up alternately. It is interesting to visualize theoretically the

blood running down the channels and dilating them when the head is up and the vessels emptying out and contracting when the head is down. Theoretically, this is a way of exercising one's blood vessels without working. Actually, there is no evidence that this is true, and actually there is almost no evidence that this method of treatment improves the peripheral arterial circulation, but in actual clinical practice, there is a good deal of evidence of benefit from this method of treatment.

Intermittent venous occlusion has had a great deal of vogue in the last two or three years, and physiologists have debated bitterly whether or not this method of treatment improves the arterial circulation. The cuff is inflated by a piece of machinery to a pressure of about 80 mm. of mercury and is kept inflated about four, five, or six, or even as long as nine minutes to impair the venous return, and then the cuff is deflated for a period of about two minutes. During the period of deflation of the cuff, there is marked increase in the arterial circulation, but the evidence now suggests that this improvement in the arterial circulation which occurs during the period of deflation of the cuff is nothing more than a repayment of the deficient circulation which occurred during the period of inflation of the cuff. Recent studies also indicate that this method of treatment does not produce any permanent improvement in the arterial circulation, but some of those who are interested in this mechanical method of treatment agree that the clinical results from it are occasionally rather satisfactory.

All of you are familiar with the glass boot which was in the headlines about 1934 under the heading, "New feet for old," and similar titles. I fear that no one has got new feet for old as a result of this method of treatment. What I have said about intermittent venous occlusion is almost entirely applicable to intermittent suction and pressure. There is a tendency for almost everyone to move this method of treatment a little closer to the attic each year. That is not said facetiously, because it is true that the method has not

lived up in any degree to advance notices relative to the benefit which results from it. However, one occasionally sees rather good results from this method of treatment and my colleagues and I continue to use it.

Intravenous injections of hypertonic solutions of sodium chloride have a good deal of vogue, particularly in New York where Dr. Silbert of Mt. Sinai Hospital has had such a large experience. In the hands of many others, the method has not proved beneficial, and it is one of those methods of treatment for which I cannot offer you any good scientific evaluation. My own opinion is that it is not this method of treatment which improves the condition of patients but it is rather the meticulous care which those who use it give their patients in other regards. There is an honest difference of opinion relative to this.

Diathermy will also improve the arterial circulation substantially, but it is well to keep in mind that diathermy or heat of any kind exceeding 95° F. should not be applied to the extremities themselves. One gets just as good effect on the circulation by applying the terminals to the back and abdomen and then one does not have as much danger of burns. There is a substantial increase in the temperature of the skin of the extremities as a result of diathermy, and this increase in the temperature of the skin is a reliable indication of improved arterial circulation. The disadvantages of this method of treatment are that the effects are of short duration.

The method of active treatment which I like the best in dealing with thrombo-angiitis obliterans is the use of typhoid vaccine. After each intravenous injection of typhoid vaccine, the circulation is improved for a variable period but the beneficial effects of this type of treatment frequently exceed in duration the persistence of the fever and of the improved arterial circulation. Most of us believe that this method of treatment has a definite deterrent effect on the disease itself.

My colleagues and I are studying a heating unit which we have devised in connection with Mr. Gayne of the Colvinex

Corporation. It is an offshoot of the electrically heated underwear that deep-sea divers use, and of the electrical units which heat the airplane motors in cold climates. It is a thermostatically controlled unit, a sleeve, which is very simple of application. It puts heat into the body, and the body, in an attempt to relieve itself of this additional heat, responds with marked increase of the circulation in the extremities.

There are a few difficulties in this method of treatment and the mechanical factors have not all been worked out. The method has the advantage that it is simple to apply and that it can be used over long periods. There is marked improvement in the peripheral circulation as a result of this method of treatment.

Tissue extract has only one function in the treatment of impaired arterial circulation: that of increasing the distance patients can walk before they get the pain of intermittent claudication. Alcohol is a very good vasodilator. One needs to watch, of course, that patients do not become habituated to the use of alcohol, which is very easy when it is prescribed for patients who have chronic diseases. High environmental temperature also improves the circulation so that in our hospital unit each room is so arranged that the temperature may be controlled at 90° F. Sympathectomy also has very distinct value in the treatment of impaired arterial circulation. It is a strange commentary on the wise provisions of nature that an extremity which is dying for lack of blood has within itself the potentialities of greatly increasing the arterial circulation, yet does not do so. In all the chronic occlusive arterial diseases there is, in addition to the organic changes in the arteries, a degree of arteriolar spasm which further impairs the arterial circulation. This arteriolar spasm may be relieved by sympathectomy, which may substantially increase the circulation. Sympathectomy is a method of treatment in thrombo-angiitis obliterans which is particularly good, but is used only for

those patients who do not have gangrenous lesions and who are free of pain.

The most important thing in the treatment of thrombo–angiitis obliterans and arteriosclerosis obliterans is a feeling of optimism. There is too much pessimism; too many physicians feel, "Well, there is nothing to do about this condition; we might just as well give up." However actual clinical experience shows that much can be done to rehabilitate these patients, to preserve their extremities and to allow them to carry on with fairly normal activity. The relief of pain is always a great problem and the use of acetylsalicylic acid and codeine is usually a reasonably good method of control of pain, but sometimes morphine is necessary. Again one should guard against habituation. Cobra venom, which had considerable vogue in the control of pain of cancer, in my experience has not been of any great value, if of any value, in the relief of pain in chronic occlusive arterial diseases. Occasionally the peripheral nerves may be sectioned and resutured, or alcohol may be injected into them to produce anesthesia of the peripheral parts, and sometimes this gives startling relief of pain without impairing motor function.

There is always the possibility that amputation will be necessary, but amputation should be reserved until one is entirely convinced that more conservative methods of treatment will not produce improvement. In thrombo–angiitis obliterans, amputation need not be carried out in more than 10 per cent of instances, if the disease is treated adequately. The percentage is a little higher in arteriosclerosis obliterans, and the problem in arteriosclerosis is somewhat different, because people who have arteriosclerosis obliterans also have diffuse arterial disease which will terminate fatally, and the problem is one of preserving the extremities until some other event brings this unwished for termination. Amputation of fingers can be performed satisfactorily; healing almost always occurs, while amputation of toes can rarely be perform-

ed satisfactorily, probably in not more than 5 per cent of instances in which amputation of fingers is done.

In conclusion I should like to emphasize again that adequate medical treatment will accomplish a great deal in thrombo-angiitis obliterans and arteriosclerosis obliterans and will do a great deal to prevent patients who have these diseases from becoming chronic invalids. Early recognition and intelligent treatment in which the internist, orthopedist and neurosurgeon co-operate are desirable.

PRESIDENT CRAGIN—We have heard Dr. Allen's very lucid description of the various phases of peripheral artery disease, especially in two instances, and I am very sure that Dr. Allen will be very glad to answer any questions that may be propounded to him. Has anybody anything to offer?

DR. SCHWANTZ—I should like to ask a question. These people that get gangrene are generally older people, and very frequently they also have extensive arterial diseases involving the heart, let's say. In that case, what effect has the typhoid vaccine therapy on the condition? Can they stand it?

DR. MCNORTON—I am interested in knowing why you keep the heat at 90°.

DR. WILSON—I should like to know the effect of this disease upon the healing and handling of fractures.

DR. JAN NYBOER—One of the biggest problems I think we have in following these diseases is the quantitative method by which we can determine the volume flow through the extremities. Naturally, the mechanical approach has been very difficult. At Post Graduate Hospital in the last few years, we were working on a theory that we might be able to use a radio frequency current and pass it through the various extremities, and detect it again and use this electrical pulsation as an index of the velocity and of the volume flow.

Dr. Irving S. Wright and Dr. Canterbell and myself have made some success in this direction. Apparently the difficulties that one has in quantitating the mechanical methods arises in the electrical methods. I actually believe we will be able to quantitate the flow in the upper extremities with fair

success, but due to the irregularly shaped toes and the difficulty in applying electrodes accurately, and measurement of the volume of the lower extremities, we are going to meet with failure in the lower extremities, and if one follows the literature in the next few years, one would have to follow the measurements in electrical impedance. These curves are reproduced on a photographic strip of film similar to an electrocardiograph.

DR. ARMSTRONG—May I ask for a little more information about the explanation of this condition in young people? In my own experience years ago, it occurred in quite young people, even in a boy of 12, necessitating amputation.

DR. PARKS—I should like to ask Dr. Allen how important he thinks the control of diabetes is in the prevention and treatment of those cases of peripheral arteriosclerosis associated with that disease.

DR. LEE—Do you recommend the use of alcohol by people past 40?

DR. ALLEN—Typhoid vaccine I think should be used cautiously in patients who have arteriosclerosis, and certainly I would be very reluctant to use it in a patient who had clinical manifestations of impairment of his coronary artery circulation.

The question of heat can be very simply answered in about the following manner: If one applies heat locally, there is a tremendous stimulation to the improvement of the arterial circulation. As a matter of fact, the best way of improving the arterial circulation is to apply heat to it locally. However, if this heat exceeds 95, and some men say 90, but 95° Fahrenheit, it increases the metabolism of the tissue so that the beneficial effects of improvement of the circulation are offset by increased metabolism. Cessation of pain claudication is indeed an indication of improved circulation.

I wish I could answer the question about fractures and healing. I don't know the answer, and I don't know what to say except that I have seen three individuals who had fractures with marked impairment of their arterial circulation who got

prompt healing of their fractures. I don't think that would happen routinely, but just in the three cases that have come to my attention, that has happened.

It is indeed a difficult method to quantitate the peripheral arterial circulation and to determine changes in the peripheral arterial circulation, and I am very much interested in Dr. Nyboer's comments on this. The best and simplest way which we have now is to put the patients in a room of about 90° Fahrenheit and see what the maximum temperature of the skin reaches as a result of being in this environment.

Young people do not get thrombo-angiitis obliterans. The youngest patient which we have seen I think is 16. I think he had had his disease two or three years. When commenting on thrombo-angiitis obliterans, I said that there were two things which were important, and one was the use of tobacco. Do you remember whether your patient smoked at a young age, Doctor?

DR. ARMSTRONG—I have particularly in mind a boy I first saw at about age 15 who came into the hospital with redness of both feet running about half way up the legs. He was sent to some clinic. Then the next time I saw him, he had had an amputation of first one leg and then later on the second, and the last time he came in, it was an emergency of thrombosis. I think he couldn't have been more than 19 at that time, and he died.

DR. ALLEN—He might have been a heavy smoker. The youngsters we have seen with the disease are heavy smokers. It is an inflammatory disease of the arteries, and the surprising thing is it doesn't affect younger individuals more than it does. It is very rare below the age of 20.

The control of diabetes is important, and I am not going to say much about that, because I suspect that later on in the program Dr. Root or someone will comment upon that.

I don't know what to say about this alcohol in individuals past 40, because out in Minnesota none of us uses alcohol, and I am not yet 40, and I have no personal experiences to

offer, but I should say that if alcohol is good for anyone, it ought to be good for individuals past 40.

PRESIDENT CRAGIN—Thank you very much, Dr. Allen. I am sure you set a great many uneasy consciences at rest.

Diabetes is an old disease, known at the time of Census. I occasionally read the Bible. I wonder whether Job didn't have diabetes, because I remember he was a heavy eater before he started in having boils. Any Bible student might carry that through if he wished to.

It is always well to have an occasional paper on diseases which seem to be increasing in frequency, and to bring up to date recent developments along these lines. Whenever we wish to get a combination of clinical and insurance experience, we turn to Philadelphia, and the Penn Mutual Life Insurance Company, as there is found a man who has appeared before this Association in previous years. He is a graduate of Princeton, received his medical degree from Harvard, is a Fellow of the American College of Physicians, an Associate Professor of Metabolic Diseases in the University of Pennsylvania, Graduate School, and author of numerous articles on diabetes.

May I present Dr. Edward S. Dillon, Assistant Medical Director of the Penn Mutual Insurance Company, who will bring us up to date in "Recent Developments in Diabetes." Dr. Dillon!

RECENT DEVELOPMENTS IN DIABETES

By Edward S. Dillon, M. D.

Assistant Medical Director
The Penn Mutual Life Insurance Company,
Philadelphia, Pa.

Not infrequently, we read and hear that hyperglycemia and glycosuria, provided there is no ketosis or dehydration, do not seriously interfere with the good health of diabetic patients. It is pointed out that, in the presence of hyperglycemia, infections do not seem to be more common and surgical wounds seem to heal as well as at normal blood sugar levels. Certain clinics have advocated the use of especially liberal diets, on the theory of stimulating the endogenous production of insulin by the remaining functional islet cells of the pancreas.

All of this, it seems to me, constitutes dangerous doctrine for the routine management of diabetic patients. The principles of undernutrition enunciated by Allen prior to the discovery of insulin, and the principles of control of the diabetes, are just as valid as ever; and recent animal experiments, to be discussed below, add support to these principles.

That most adult diabetics are overweight before the diabetes begins is a well known fact. Approximately 80% have been 5% or more overweight. The likelihood of developing diabetes increases rapidly with the degree of obesity. For men past age 45, the diabetic mortality is low for those of normal weight, doubles for those five to 14% overweight, is four times as great for those 15 to 24% overweight, and is ten times as great for those over 25% overweight. To avoid diabetes, avoid obesity. Overweight is equally to be avoided after the diabetes has developed. In a given diabetic, the diabetes is mildest and best controlled when he keeps his weight at normal or slightly below. The most important single feature about a prescribed diet for a given diabetic is that the calories be such as will maintain his weight at the desired level. Whether the fat be relatively high and the carbohydrate low, or vice versa, is

probably not so very important. For adults of normal weight, few clinics now prescribe diets containg less than 100 grams of carbohydrate and, usually, not more than 200 grams.

Control of the diabetes every day, all day, must be insisted upon. To mention only one of the complications of diabetes, gangrene—the thing which has impressed me most is that, usually, gangrene does not occur in severe diabetics, but in mild cases which have been uncontrolled over a period of years. The usual story when a diabetic is admitted to a hospital with gangrene is (a) that he did not know he had diabetes, (b) that sugar had been found in the urine some years before and he had not followed instructions, or (c) that, when the sugar had been found, his physician had given no instructions other than to cut down on sugar and starches.

The diabetes is controlled (a) when the patient is on a diet properly proportioned in protein, fat, and carbohydrate, containing sufficient minerals and vitamins, and having the right amount of calories to maintain his weight at the desired level, and (b) when his insulin doses are so adjusted that, throughout the 24 hours, the blood sugar level neither falls to hypoglycemic levels nor rises above the renal threshold, causing sugar to be lost in the urine (approximately 80 to 180 mgs.). The blood sugar level is likely to be within these limits if (a) the fasting blood sugar is at a desired level, (b) there are no shocks, and (c) there is no sugar in the 24-hour urine.

In the vast majority of diabetics, the foregoing criteria can be fulfilled if careful study is given to each case; but, in a small percentage of cases, they are difficult of accomplishment. These are the cases that are sensitive to insulin. The blood sugar levels change rapidly, and the same dose of insulin on successive days may cause widely fluctuating responses in the blood sugar levels. The consequence is that these patients frequently get into shocks or spill large amounts of sugar in the urine, even get into coma. These cases are usually young, but may be of any age. Three examples, from the Philadelphia General Hospital, are presented:

CASE 1. A.K., male 31. Diet P 90, F 105, C210, and C 20 at bedtime.

4/6/41		Blood Sugar	Urine Sugar
Insulin 22P 10R	7 a.m.	192	
Breakfast	7.30 a.m.		
	9 a.m.	185	2.8 gms.
	11 a.m.	172	
Lunch	11.30 a.m.		
	1 p.m.	270	
Insulin 6R	4 p.m.	256	21.6 "
Supper	4.30 p.m.		
	6 p.m.	280	
Evening CHO	9 p.m.	228	12.0 "
	12 p.m.	156	
	3 a.m.	158	1.1 "
	7 a.m.	166	
			37.5 "

CASE 2. C.T., male, 70. Diet P 80, F 95, C 190.

6/27/41			
Insulin 25P 15R	7 a.m.	79	
Breakfast	7.30 a.m.		
	10 a.m.	88	0
Lunch	11.30 a.m.		
	1 p.m.	202	
Insulin 10R	4 p.m.	210	0.5 "
Supper	4.30 p.m.		
	7 p.m.	114	
	9 p.m.	62	1.0 "
	12 p.m.	57	
	4 a.m.	36	0
	7 a.m.	92	

CASE 3. W.M., male, 12. Admitted in coma, 7/9/41. B.S. 680, CO^2 18. Wt. 73. Standard wt. 84. Diet P 70, F 100, C 250; plus P 12, F 15, C 30, at 9.00 p.m.

8/15/41			
Insulin 18P 6R	7 a.m.	226	
Breakfast	7.30 a.m.		
	9 a.m.	280	+ + +
	11 a.m.	174	
Lunch	11.30 a.m.		
	1 p.m.	330	
Insulin 3R	4 p.m.	280	+ + + +
Supper	4.30 p.m.		
	6 p.m.	370	
Evening meal	9 p.m.	228	+ + + +
	12 p.m.	188	
	3 a.m.	121	+
	7 a.m.	88	

In Case 1, the blood sugar rose sharply after lunch and remained high during the afternoon and the early evening. As the fasting blood sugars were moderately high, the obvious insulin adjustment was to increase the protamine. Case 2 seemed to be well standardized until we did the blood sugar curve, which showed very low blood sugars during the night. Case 3 was a severe problem. The patient had been on the same diet and insulin for five days prior to the blood sugar curve. It will be noted that the values ranged from 370 to 88. The most perplexing feature, however, was that the blood sugar was 266 at 7.00 a. m. on the first day, but only 88 at 7.00 a. m. on the next day. Better results were obtained later, when he was given six feedings a day, but it was impossible to prevent occasional shocks and frequent large urinary spills.

In 1937, the startling announcement was made by F. G. Young, of London[1], that permanent diabetes could be produced in dogs by the injection of large amounts of crude anterior pituitary extract. Prior to this, Houssay and others had shown that removal of the anterior lobe of the pituitary greatly increased the sensitivity of the animal to insulin, that removal of the pituitary prior to or following removal of the pancreas diminished the severity of the diabetes, and that injection of anterior pituitary extract into animals from which both the pituitary and the pancreas had been removed greatly increased the severity of the diabetes. Since Young's original paper, other investigators—particularly Best, in Toronto, and Lukens, in Philadelphia—have confirmed his finding, and all have made further observations. The amount of anterior pituitary extract which must be injected into dogs to produce diabetes, and the time over which it must be injected, vary widely in individual dogs. Lukens[2], working with dogs, found the injection period to be from 14 to 98 days. In cats and rats, diabetes cannot be produced unless, prior to the period of injecting anterior pituitary extract, there is removed an amount of pancreas which of itself is insufficient to produce diabetes. There are, therefore, marked differences in response, both between different species and between individual

animals of the same species.

In all cases, changes are found in the beta cells of the islands of Langerhans. After the diabetes has begun, there is hydropic degeneration of the beta cells. At this stage, the process is reversible; and, with appropriate treatment, the cells may return to normal. Without treatment, however, the diabetes produced by the injection of anterior pituitary extract progressively becomes worse, long after the injections have been discontinued. The beta cells become completely atrophied, only alpha cells remaining. These changes can be observed only with special stains and fresh tissue. The islands of Langerhans of post-mortem material, stained with hematoxylin and eosin, may appear entirely normal.

If, during the period when pituitary extract is being injected, a test dose of insulin is given, the normal response to insulin does not occur[2]. On the other hand, if large doses of protamine insulin are injected daily during the period that pituitary extract is being injected, diabetes does not develop, nor does hydropic degeneration of the beta cells of the islands occur.

After diabetes has developed and the pituitary extract has been stopped, there is a normal response to a test dose of insulin, and treatment with insulin stops the tendency of the diabetes to become continuously more severe. If the beta cells of the islands have atrophied, treatment with insulin cannot restore the function of the islands or cure the diabetes. If, however, at the time when treatment with insulin is started, the beta cells of the islands have not progressed beyond the stage of hydropic degeneration, then the beta cells may return to normal, accompanied by permanent cure of the diabetes.

Best and his coworkers[3] found that fasting or fat feeding tends to prevent the rapid fall in the insulin content of the pancreas and the degenerative changes in the islet cells resulting from daily administration of anterior pituitary extract.

In the condition which results from the injection of anterior pituitary extract, it would appear that there is an excessive

demand for insulin, resulting in functional strain of the islet cells and, finally, in their degeneration. There is no evidence that this result is due to a primary degenerative effect of the extract, exerted directly on the islet cells.

What are the lessons for human diabetics in these discoveries on animals? Joslin and his associates[4] have recently reviewed their cases and conclude that, although no cure of diabetes can be claimed, early vigorous treatment, with either a restricted diet or insulin or both, in certain patients, seems in retrospect to have brought about an amelioration in the diabetic condition of far greater degree than one would anticipate.

Rest the islands! This is done when a diabetic reduces his obesity, when he keeps his blood sugar level at a normal level every day, all day. We see the return of islet function when an uncontrolled diabetic comes under treatment. To control his diabetes, large daily doses may be necessary, say 80 units. After the diabetes has been under control a short time, the insulin must be lowered, perhaps to 40 units, perhaps lower, because the islets have recovered some of their lost function.

For many years, it has been stated as fact that fatty acids break down two carbon atoms at a time (i. e., successive beta oxidation) and that fats can burn only in the fires of carbohydrates (i. e., that the ketone bodies can be oxidized only when they are in a definite quantitative chemical relation with glucose or certain of its products). Stadie[5], in a series of brilliant papers, has recently proved that both of these beliefs are false. His conclusions are that in the liver, by a process of multiple alternate oxidation, four ketone bodies are produced from each molecule of fatty acid, that both ketone bodies and fatty acids can be utilized in the periphery, that all ketone bodies will be utilized if the total fat metabolism does not exceed 2.5 grams per kilogram of body weight, and that there is no correlation between the amount of ketone bodies oxidized and the amount of glucose oxidized.

Diabetes is particularly likely to develop in overweight individuals. It is also likely to run in families. It might be thought, therefore, that individuals having a family history of

TABLE I

AGE AND WEIGHT DISTRIBUTION OF 2000 APPLICANTS WITH FAMILY HISTORY OF DIABETES (FIGURES ABOVE) COMPARED WITH 2000 APPLICANTS WITHOUT FAMILY HISTORY OF DIABETES (FIGURES BELOW)

	Age 15–19	20–24	25–29	30–34	35–39	40–44	45–49	50–54	55–59	60–65	TOTAL
Weight +26% & over	0 – 0	2 – 1	8 – 3	4 – 7	6 – 7	10 – 5	7 – 7	8 – 2	2 – 1	0 – 0	47 – 33
+25% to +16%	0 – 1	3 – 13	17 – 12	16 – 15	25 – 10	33 – 11	23 – 12	17 – 5	2 – 2	1 – 2	137 – 83
+15% to +6%	3 – 8	15 – 28	46 – 54	73 – 61	63 – 58	79 – 32	45 – 28	29 – 11	11 – 1	7 – 2	371 – 283
+5% to -5%	16 – 59	81 – 168	144 – 144	162 – 126	135 – 113	130 – 70	86 – 46	47 – 25	17 – 15	8 – 6	826 – 772
-6% to -15%	28 – 97	91 – 161	108 – 120	87 – 102	58 – 47	43 – 18	24 – 11	14 – 11	6 – 4	5 – 0	464 – 571
-16% to -25%	8 – 44	35 – 71	37 – 51	28 – 38	14 – 13	8 – 8	0 – 5	5 – 0	3 – 0	0 – 0	138 – 230
-26% & less	3 – 5	3 – 13	5 – 4	2 – 4	2 – 1	1 – 0	1 – 1	0 – 0	0 – 0	0 – 0	17 – 28
TOTAL	58 – 214	230 – 455	365 – 388	372 – 353	303 – 249	304 – 144	186 – 110	120 – 54	41 – 23	21 – 10	2000 – 2000

TABLE II

COMPARISON OF RATIO OF OVERWEIGHTS BY AGE GROUPS OF APPLICANTS WITH DIABETES FAMILY HISTORY AND WITHOUT

	Weight	Ages 15–39	Ages 40–65
Diabetes Family History	Cases +6% and over	281 (21.2%)	274 (40.1%)
	Total cases	1328	672
No Diabetes History	Cases +6% and over	278 (16.8%)	121 (35.5%)
	Total cases	1659	341

diabetes would show a tendency toward overweight. I collected 2000 cases of applicants for new insurance whose parents or siblings had diabetes; and I compared them with 2000 other cases, consisting of all applicants and collected during several short periods of time. Both groups have been distributed, in Table 1, by age and weight. There were 432 female applicants in the diabetes-family-history group and 363 female applicants in the general group.

Diabetes is a disease chiefly of middle and old age. Because of this fact, as may be noted in Table I, there are relatively more of the diabetes-family-history group in the older ages, relatively fewer in the younger ages.

The weights used in Table I were taken from the case work-sheet, on which the weight is usually recorded in multiples of five. In our numerical system, standard weights are for age 37, adjustments for age being made in the numerical ratings. The diabetes-family-history group has more overweight individuals than the general group, because it has more older individuals than the general group. It becomes necessary, therefore, to compare the ratio of overweights in each quinquennium, and for this the number of cases is too few. However, if the applicants are divided into two age groups, 15-39 and 40-65, and if the number of overweights in each age group is compared with the total number in each age group, the percentage of over-

TABLE III

APPLICANTS WHOSE FATHERS, MOTHERS, BROTHERS, OR SISTERS HAVE DIABETES OR DIED FROM DIABETES

Father died	565	
Father has	227	
Mother died	525	
Mother has	334	
Brother died	121	
Brother has	56	
Sister died	89	
Sister has	43	Total 1960
Father died — Mother died	3	
Father died — Mother has	1	
Father died — Brother died	3	
Father died — Brother has	7	
Father died — Sister died	2	
Father died — Sister has	1	
Father has — Mother has	1	
Mother died — Brother died	5	
Mother died — Brother has	1	
Mother died — Sister died	2	
Mother died — Sister has	1	
Mother has — Brother has	1	
Mother has — Sister died	1	
Mother has — Sister has	1	
2 Brothers died	4	
Brother died — Sister died	2	
Brother has — Sister has	1	
2 Sisters died	1	
Brother died — 2 Sisters died	1	
Brother died — Sister died — Sister has	1	Total 40
		2000

Total Fathers — 810
" Mothers — 876
" Brothers — 207
" Sisters — 149

weights is somewhat higher for those with a family history of diabetes (Table II).

The distribution of the relatives who had diabetes is of some interest and is set forth in Table III. There were 810 fathers involved and 876 mothers. The incidence of diabetes is, of course, considerably greater in females than in males. The lack of a greater preponderance of mothers over fathers in this series is accounted for by the fact that fathers as a whole are older than mothers. In the cases where the father and the mother were both living, the fathers averaged 3.47 years older than the mothers and had that much more opportunity to develop diabetes.

In estimating the magnitude of diabetes as a public health problem, we need to know chiefly two things: First, how many diabetics there are; and, secondly, what proportion of them receives really adequate treatment or, conversely, what proportion needlessly dies or is needlessly disabled.

It is estimated that there are now about 660,000 diabetics in the United States and several times that number of persons now living who will develop the disease before they die. This estimate is made by multiplying the number reported as dying each year of diabetes by the average number of years which a diabetic lives after developing the disease. Whether the total number recorded as having died of diabetes will be an accurate index of the number which did die of the disease will depend on:

(1) Whether all diabetics are attended by a physician at death;
(2) Whether the diagnosis of diabetes is made;
(3) Whether the diagnosis of diabetes is recorded by the attending physician on the death certificate; and
(4) Whether the health authorities properly record multiple causes of death.

Items 2–3–4 all have difficulties peculiar to diabetes. Diabetes is a chronic, killing disease and differs from most other chronic, killing diseases in that it can be almost completely

controlled by adequate treatment, but is subject to numerous complications, which may be due to lack of control of the diabetes or may be independent of the degree of control.

A middle-aged or elderly diabetic usually does not die because of the immediate effects of diabetes. At the time of death, (A) the diabetes may be (a) well controlled, (b) indifferently and intermittently controlled, or (c) uncontrolled (acidosis, dehydration, malnutrition); (B) arteriosclerosis (coronaries, legs, brain, kidneys) is usually present, which (a) may have resulted from previously poorly controlled diabetes or (b) may be due simply to senile changes; and (C) an acute infection is often present, which (a) may be of such severity as to cause death of itself or (b) may make the diabetes much more severe or (c) may be only a terminal incident (bronchopneumonia) just prior to death. Often, we are sure that a diabetic would not have died as soon as he did if he had had good treatment; on the contrary, we are often certain that a diabetic was doomed to die, regardless of treatment; but, in between, there are a great many cases regarding which a sound judgment concerning inevitability of the death is difficult to make. To make such a judgment, it is necessary to have an accurate record of the entire course of the disease, as well as accurate clinical and chemical data of the last illness.

The mortality rates of diabetes vary enormously in the different states, ranging, in 1938, from 41.9, for Rhode Island, to 8.7, for New Mexico. The mortality rate is especially low in the southern states, and in the past this has been ascribed, in part at least, to the effects of climate. Items 1–2–3–4, mentioned above, probably have much more effect on the reported mortality rate of diabetes than has climate. Joslin's[6] recent experience in Arizona leads him to conclude that the incidence of diabetes is highest where (a) the average age is oldest, (b) women predominate, (c) obesity is more frequent, (d) the proportion of Jews is greatest, (e) medical supervision is closest, and (f) deaths are most accurately reported.

There should be scientific surveys of diabetes in each city and state, in order to arouse the laity to the dangers of diabetes, to create a demand for good treatment, and to put the medical profession on its toes. Last year, in each case where diabetes appeared on the death certificate, the Committee on Diabetes of the Philadelphia County Medical Society sent a detailed questionnaire to the physician or the hospital. I reviewed a large number of the replies. In spite of the fact that the questions had been drawn up with care, the data on the replies were adequate to judge only whether or not the patient had received good treatment. In cases of poor treatment, it was impossible to judge the relative shortcomings of the patient and the doctor. Very often, it was impossible to judge why the patient died and whether good treatment would have saved him. Large, scientific, worthwhile surveys not only would require intelligent direction, but would cost money.

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PRESIDENT CRAGIN—Fortunate is a man who can get as a discusser for his paper a member of the famous Joslin Clinic of Boston. Our discusser received his A. B. and M. D. from Harvard, was a Fellow at Johns Hopkins in 1920, has been physician to the New England Deaconess Hospital since

1923, Associate in Medicine at Harvard Medical School, Fellow of the American College of Physicians, a member of the A. M. A., and a member of the Clinical and Climatological Association and the American Society of Clinical Investigation. Although he doesn't seem to have his uniform on, he is a Lieutenant Commander in the Naval Reserve.

Dr. Root has kindly consented to discuss Dr. Dillon's paper. Dr. Howard F. Root, Physician of the New England Deaconess Hospital, and Associate Physician in Medicine to the Harvard Medical School. Dr. Root!

DR. ROOT—Dr. Cragin, Members of the Association, Ladies and Gentlemen. Dr. Cragin's introductions indicate that he is a student of heredity or of genetics, or something; he leaves no details out so far as our past record is concerned, except those which he is kind enough to conceal, I suspect.

It is a real pleasure for me to have this opportunity to speak before you for the first time. I am only a clinician and not a statistician. I wish I might have today presented some statistics from the big clinic in Boston. For one reason it would give me an opportunity to express the very great appreciation which all of us there feel for the courtesy and the tremendous amount of work which the two gentlemen here present, Dr. Dublin and Mr. Herbert Marks, have given these many years. Many of you know of it. This cooperation statistically in the records of Dr. Dawson's patients back to 1898 has yielded results which to us at least have been very valuable, and I think that relationship for years has set a standard perhaps, or has been an experiment which probably in the future will be very helpful in leading the insurance companies and perhaps other agencies who are interested in new efforts to improve public health, to improve longevity of which we have heard so much here that I can't help stopping at this moment to speak with the greatest appreciation of that relationship with insurance medicine.

Dr. Dillon's stimulating paper on diabetes dropped a bomb of the illuminating rather than destructive type, where

it should do some good, in the camp of those who do not encourage exactness in treating diabetes. He emphasizes again that the object of treatment is the control of disease and that the control of disease means the abolition of those deviations from normal produced by the disease.

In the latter part of the 19th century, Naunyn and VonNorden, by the use of green vegetable days showed clearly that the avoidance of overeating was fundamental in diabetic treatment. Then came the experiments of Allen who demonstrated convincingly that if the total number of calories in the diet per day were suitably restricted and adjusted to the capacity of the individual patient that progression in the severity of the diabetes could be prevented. Allen and the food scale which followed his demonstration changed diabetes in the hospital so that even before the use of insulin the development of diabetic coma in hospitals had ceased. It was clear that if diabetic coma could be prevented in the hospital it could also be prevented in the home.

Then came the discovery of insulin and an era in which not only control of the diabetes but actual improvement in diabetic severity and the conversion of severe cases into mild cases became a demonstrated fact. I cannot but reiterate Dr. Dillon's excellent statement that the control of the diabetes every day all day must be insisted upon. It is usual to think of five different aspects of carbohydrate metabolism which must be controlled if proper regulation of the diabetes is to be maintained. The first of these is acidosis due to ketosis; second is the adequacy of the diet, both in respect to total calories but in content of vitamins, minerals and in proportion of carbohydrate, protein and fat; the third is the blood sugar already emphasized by Dr. Dillon; four is the efficiency of the action of insulin; and the fifth is the respiratory quotient relationship, that is, the respiratory quotient should be maintained as nearly normal as possible. In the control of the blood sugar Dr. Dillon has cited cases of insulin sensitivity and they constitute an important group.

There is another group of patients whom we are destined to see more frequently in the future than in the past, namely, those with insulin resistance in whom the action of insulin is less efficient. Here I refer to the fact that occasionally a diabetic patient will, rather quickly or sometimes gradually, develop a state in which his previous dose of 40 or 50 units of insulin a day is ineffective and it is then found somewhat to the physician's surprise that doses of from 100 to 500 or even 1,000 units a day are required. When such a state of resistance comes on in a short period of time diabetic coma may supervene within a few days unless the patient has been properly advised to increase his dose of insulin rapidly. Indeed, I have seen a young clergyman in this condition who, while receiving 400 units of insulin a day, went into diabetic coma in the hospital which was so severe that he required 2,000 units in 24 hours to bring him out of coma. During the next few months his insulin requirement ranged from 1000 to 1500 units a day. Yet, at the end of the year this phase of insulin resistance had subsided and he was back again to his previous requirement of about 40 units. Various causes for this state of insulin resistance have been observed. The first one is disorder of the glycogenetic function of the liver associated with such conditions as hemochromotosis, or obstructive jaundice. One of our patients, a woman of 60 years, had intermittent jaundice for 6 months. At operation a stone in the common duct was removed. She required 1,000 units a day for a brief period about the time of her operation but during the next few months the insulin requirement rapidly fell and now at the end of 18 months she is sugar free and has a normal blood sugar without any insulin whatever. The second cause of high insulin resistance is found in the active stages of acromegaly and in hyperthyroidism. A third cause is disorders of the skin where the function of the skin as a storehouse for glycogen is disturbed. The fifth is acute infection. In one case of pneumonia, mild diabetes became so severe that the insulin requirement rose to 200 units and yet after recovery

from pneumonia the insulin requirement fell to zero. In dealing with surgical infections, also the action of insulin is undoubtedly responsible in considerable measure for the great improvement in mortality rates among diabetics. It seems quite possible, from the experiments of Young, Best and Lukens that a mild diabetic patient under the influence of severe infection may undergo severe and acute toxic damage to the Islands of Langerhans which will become permanent unless the patient during this acute stage receives a large supply of insulin which will take the burden off the damaged islands. Then when the acute stage is over regeneration may be complete. Insulin resistance seems to occur in some patients with a cyclic character which probably suggest the well-known cyclic variation in the intensity of acromegaly. Indeed, one recent patient, a woman of 72 years, took 185 units a day for a year. During this period the Ascheim-Zondek test was positive. Then rather suddenly this phase of resistance disappeared and insulin could be withdrawn entirely. Now the A–Z test is negative. This has also been true of two other patients. These three patients suggest the existence of periods of pituitary overactivity unlike acromegaly but with a definite effect upon the insulin requirement.

These instances of insulin resistance where the requirement rises to from 100 to 300 units will probably be somewhat more frequent in the future as the number of patients with diabetes of long duration increases. They are by no means rare in people seventy years and over, but naturally much less frequent than in the age period from thirty years to fifty, and curious enough, so far I cannot recall any case of high degree of insulin resistance (200 units) reported in children under 20 years of age.

The metabolic study of the last 12 extraordinary cases of insulin resistance has explained some cases, but not all. The important observation is that insulin always works. So far we have found no case of insulin resistance even where a dose of 2000 units or more has been required in whom we

could not reduce the high blood sugar to normal and in whom we could not raise the respiratory quotient from a low level to a normal level if we gave sufficient insulin. It can be said that insulin always acts and the required control of the blood sugar and respiratory quotient can always be brought about if the proper dose of insulin is used in conjunction with the proper diet.

Indeed, these experiments go a little farther in answering questions which have to do with the old problem of the nature of diabetes. There has for many years been a dispute between those who maintain that diabetes is primarily due to an inability to utilize and oxidize glucose, and those who maintain that diabetes is essentially due to the overproduction of glucose from glycogen and from protein and fat in the liver. It has been debated whether the action of insulin is really to promote carbohydrate oxidation or merely to check the breakdown of glycogen into glucose and to check the formation of glucose from protein and fat. If the action of insulin were only to promote glycogen formation from glucose and to prevent the breakdown of glycogen into glucose, then it should be possible when you give glucose by mouth or by vein to find such a dose of insulin as would convert that glucose into glycogen and not cause any change in carbohydrate oxidation. Under these circumstances, the rise in the respiratory quotient following the giving of the glucose would not occur. Actually, however, in all our experiments even with the most resistant cases, when we gave glucose solution by mouth or by vein, if a sufficient dose of insulin is given you always obtain the rise in respiratory quotient which is characteristic of administration of glucose in the normal, and which is then interpreted as due to an increase in the proportion of carbohydrate in the metabolic mixture which is being oxidized. The action of insulin, therefore, in all our patients no matter how severe, has been to bring about a rise in respiratory quotient. It is possible that this rise in quotient is due to conversion of carbohydrate into fat, and then that the fat is utilized as fat. Indeed, some experimental data

would confirm this. However, this pathway still means that insulin increases carbohydrate utilization, no matter what the intermediate steps may be. On the other hand, it is certainly true that under the condition of McLeod's experiments the administration of insulin to depancreatized animals always increased the amount of glycogen deposited and also raised the respiratory quotient. Actually, there is usually a fall in the amount of fat deposited in the liver under those circumstances rather than an increase. The evidence, on the whole, tends to support the belief that a rise in respiratory quotient obtained when insulin is given is due to an increase in the proportion of carbohydrate being actually utilized whether by direct oxidation or by preliminary transformation to fat.

Another extraordinarily important effect of insulin is shown in the experiments of Best, Lukens and Young to which Dr. Dillon has referred. It is clearly evident that one specific function of insulin is the protection of the Islands of Langerhans so that under favorable circumstances their ability regenerate after injury is made possible. When diabetes is produced in animals by the injection of this crude extract of the anterior pituitary gland hydropic degeneration of the beta cells which is reversible is the outstanding feature. The important point is that treatments must be begun early. Without treatment the diabetes progresses steadily and becomes permanent. I want to emphasize one point which he brought out; namely, that when the beta cells become completely atrophied and only alpha cells remain, the changes can be observed only with special stains in fresh tissues. In postmortem material stained with the ordinary stain, the Islands of Langerhans may appear normal. This fact has undoubtedly misled many pathologists not especially interested in diabetes into describing the pancreas of diabetic patients as being normal, when in reality had the postmortem material actually been stained with proper stains and soon after death, very definite changes would have been evident. Clearly,

much is yet to be learned about the pathology of the pancreas and what is needed is not a report of a few score autopsy examinations, but thousands of autopsy studies of diabetic patients carried out in all parts of the country.

The independence of the pancreas is extraordinary. One might be led to jump at the conclusion from these experiments that it is probable that some type of pituitary influence has been the active agent leading to the degenerative changes in the Islands of Langerhans in all cases of diabetes. As yet, no data definitely support this conclusion. Actually, Best and Haist have shown that in animals from whom the pituitary has been completely removed, the same variation in insulin content of the pancreas can be produced by variations in diet. The insulin content of the pancreas is not affected by removal of the pituitary gland. Indeed, within wide limits the normal pancreas can regulate insulin production in response to dietary needs in the complete absence of the pituitary.

Although we hesitate to use the word "cure" in diabetes, there can be no doubt that instances of remission in diabetes for periods, often years in length, are becoming by no means rare. Two young women in our experience have taken insulin for the greater part of their childhood and now in their twenties, one has been able to go without insulin for five years, with practically an unlimited diet, and the other has reduced her insulin dose to a matter of four or five units and even that could be omitted. It is true however, that the instances of remission are more frequent in middle life, a fact which is not difficult to understand when we realize that from middle life onward the basal metabolism slowly but steadily declines and the general level of metabolic and particularly glandular activity must decline. Two men whom I have seen recently are now in their middle fifties. Each had more than seven percent sugar in the urine and high blood sugar levels, one case six years ago and in the other

eight years ago. One of them had a carbuncle at the time when the sugar was present. Both took insulin for a period of some months, yet neither has required insulin, nor has required any great restriction of diet for the period which has now extended for six years in one case and eight years in the other. Remissions do occur under the influence of certain types of liver disease, pituitary disease and notably patients, as in the man with the carbuncle, who have had infections such as pneumonia in an early stage of the disease and have by reason of the infection required large amounts of insulin. In certain cases, the use of large doses of insulin has seemed to be followed during the months or years thereafter, by a remission in the diabetes. Dr. Dillon's statistics are most interesting. The fact that among the families of his insurance patients, diabetes was known in a higher number of cases among the fathers and mothers may really have reflected only the difference in the medical care at an earlier stage. Another possibility occurred to me as an explanation for the well-known fact that in the present day mortality tables, many more women seem to die with diabetes than men. Is it not possible that since men were formerly insured rather more frequently than women, particularly for larger amounts, that diabetes might be concealed on the death certificate rather more frequently than among women. However, the striking fact to me is that there is not a very great difference in the frequency of obesity among insurance candidates who did not know of diabetes in the family. One must always question the reliability of family histories and I know that Dr. Dillon has taken this into account. But, his figures do raise the interesting question again as to what is the relation of obesity and heredity to diabetes. There can be no question about the great frequency of hereditary diabetes in the family of the patients with diabetes. On the other hand, as studies are made of the families of people without diabetes, it is quite surprising to find what a high incidence of diabetes there is in their families also, especial-

ly if individuals whose intelligence, education and interests are likely to make them seek knowledge of their families' health. For example, Dr. Joslin made careful inquiry among medical audiences at one time including medical students and physicians. Among such audiences and among nurses and dietitians, all of whom may be assumed to have a rather more intelligent interest in the health of their relatives, higher percentages of diabetes in the family were found than occur in ordinary diabetic groups. It is difficult to avoid the conclusions that whereas a relatively small percentage of the community have or will get diabetes, a much larger percentage of people in the community do have diabetes in the family and probably do carry the hereditary tendency. This makes the importance of these precipitating factors relatively much greater. The study of obesity therefore and of infections which may involve the pancreas and particularly of those endocrine complications as well of the dietary physical habits of the individual must be pursued farther.

The change in character of the diabetic population is well demonstrated, first in the increasing duration of life of the diabetic patient and in the change in vulnerability of a diabetic. Thus, the mortality rate for tuberculosis of diabetic patients has fallen rapidly in the last twenty-five years. For some reason, the susceptibility of the diabetic patient to sepsis and particularly to septicemia seems to have changed. Fifteen years ago we were constantly on guard for septicemia, particularly in infectious lesions of the feet and now the last five years at the Deaconess Hospital we have not more than two patients with such septicemia and one is recovering with sulfadiazine. Kidney infection within the last five years has changed from a highly malignant type to a relatively mild infection. Indeed, during the last two years we have had no deaths from renal abscesses and septicemia arising in such infection. In the years preceeding that time this was a fairly important complication of diabetes. The recognition and treatment of the cyclic phase of resistance

has saved lives. This improvement in the vulnerability of a diabetic will almost surely affect insurability. Many factors have contributed to the increasing safety of the diabetic. In the future further advances and particularly new forms of insulin, such as Histone, Globin insulin, may prove valuable additions to our armamentarium.

PRESIDENT CRAGIN—Thank you Dr. Root for your discussion. It was greatly appreciated. Our next discusser is a man who is well known to us all. He has been with the Association for years. He is quite modern and up to date in most of his work, but once in a while he does slip into the old-fashioned methods. For example, last night I saw him taking the old Allen treatment for diabetes.

I take pleasure in calling on Dr. William Muhlberg.

DR. MUHLBERG—It would appear from mortality reports that diabetes is growing progressively more prominent as a cause of death. Whether this is really true is controversial. The shift in the general age distribution, the fact that diabetes has been given precedence over other reportable diseases in recent classifications, and the enhanced interest in the disease by the medical profession, may account for the successive increase in the mortality rate. In spite of these influences, most specialists incline to the belief that the disease is more prevalent and the deaths more numerous notwithstanding the brilliant advances in treatment.

The achievements of the new therapy have, however, complicated the control of the disease, because it must be confessed that the technique of modern treatment is not simple and for this reason many general practitioners are failing to avail themselves of its advantages. This is due not so much to the fact that it is beyond the skill of the general practitioner, but that he lacks the training. In Cincinnati, for instance, a careful investigation of 183 persons (out of a total of 197) dying of diabetes within a period of one year in 1939, showed the following somewhat startling results:

Insulin Dosage	No. of Cases	% of Total
Insulin used regularly	39	21.3
Insulin used only during final illness	40	21.9
Insulin used irregularly without weighed or measured diet	6	3.3
No insulin used at any time	57	31.1
No mention of insulin in record	32	17.5
Patient refused to take insulin	4	2.2
Oral tablets or capules prescribed by physician	5	2.7
	183	100.0

It is quite obvious, if our experience in Cincinnati is a criterion, that fewer than one half of diabetic patients are receiving adequate medical treatment. This is not due to a lack of faith that physicians have in the efficacy of insulin and controlled diet, but rather to the meager opportunities that they have in acquiring the skill. Furthermore, the general practitioner who does not command the facilities of a modern laboratory, is distinctly handicapped through lack of blood sugar controls, quantitative urine sugar estimations, etc. Where the patient is on the borderline of the indigent group, the problem becomes more complicated and every possible aid must be extended by diagnostic clinics.

It was in an endeavor to offer some solution of this problem that we organized in Cincinnati, about five years ago, a local Diabetes Council. About a dozen physicians, some diabetes specialists, others general practitioners, interested in diabetes, constitute the membership. The Council meets once a month at a luncheon to discuss its activities. Financially, the Council has been supported by donations made by the Western and Southern Life Insurance Company and the Union Central Life Insurance Company. A resumé of the 1940 Annual Report submitted by the President of the Council, Dr. Louis Owens, may be of interest to you. Committees

are appointed, each with a chairman, who is responsible for carrying out a definite program.

1. Committee on Publicity

Cards and placards were printed for display in doctors' offices, street cars and busses, and factories. Two radio talks were delivered, and the newspapers published 77 inches of publicity.

2. Committee on Diabetes Instruction

At the Out Patient Department of the Cincinnati General Hospital, there is a diabetes clinic, conducted by members of the Diabetes Council. Private physicians are encouraged to send their diabetic patients to this clinic for laboratory and blood sugar tests, estimation of diet requirements, administration of insulin, and general care. The patients are returned to the private physicians with a report of all findings and with suggestions for management of the patients. No charge, of course, is made for this service. If the patient is indigent, the general practitioner usually turns the patient over to the clinic. Our local Academy of Medicine has approved the plan and the general practitioners are enthusiastically in favor of it.

3. Committee for Refresher Course for Physicians

The Diabetes Council sponsored, with the co-operation of the College of Medicine of the University of Cincinnati, a refresher course in diabetes for practicing physicians. A series of 8 sessions of 2 hours each were given. Dietitians, interns and housemen, and laboratory technicians were invited to take the course. Thirty-six physicians attended, and in view of the fact that this was the first year the plan was tried, the attendance was surprisingly large. A charge of $2.00 was made for the 8 sessions.

4. Committee for Refresher Course for Nurses

In co-operation with the Ohio State Nurses Association, a refresher course for nurses was arranged in a series of 6

sessions. Eighty-six graduate nurses and dietitians registered. Since the nurse plays so important a part in the control and treatment of diabetes, the value of such courses is obvious.

5. Committee for Annual Diabetes Program

Annually since its foundation, the Diabetes Council has invited some nationally known specialist in diabetes to visit Cincinnati, at the Council's expense. The guest this year was Dr. Frank Allen of Boston, who in the evening delivered an address to the members of the Academy of Medicine. An exhibit prepared by the Metropolitan Life Insurance Company was displayed in the auditorium of the Academy and pamphlets, elaborating upon the material in the exhibit were distributed.

6. The Cincinnati Diabetes Council has taken an active part in organizing the American Diabetes Association, its first President being likewise the first Chairman of the Cincinnati Diabetes Council—Dr. Cecil Striker.

7. Additional Activities

The Council purchases from the New York Diabetes Association and distributes to all members of the Academy of Medicine their series of leaflets on the management of diabetes. The material is mailed with notices of Academy meetings.

An investigation was made of the adequacy of diabetes therapy at the Hamilton County Tuberculosis Hospital.

Efforts were made to combat the activities of a company producing and advertising a so-called cure for diabetes.

Many things remain for future programs, such as the establishment of summer camps for diabetic children, a rotating nursing service for diabetic patients that need supervision, possibly the formation of lay diabetes associations, and further encouragement and promotion of local research in diabetes as exemplified by the work done at the Cincinnati Jewish Hospital by a member of our council—Dr. I. Arthur Mirsky.

There can be no doubt that our local Council has done much to stimulate interest in both physicians and laymen.

We are hearing more and more about geriatrics these days. The span of life has reached an expectancy for new born children of over 60 years, but it is conceded that this remarkable record has been achieved almost solely through the improvements in mortality in infants, adolescents, and young persons up to the ages of 40 or 50. At the older ages the death rate is not materially different from that of 40 years ago. Except for pneumonia, affections of older ages—cardiovascular diseases and cancer—continue to exact their usual toll, because it must be confessed that our methods of prevention are not effectual and the modes of treatment not highly successful. Diabetes, a disease of the older ages, however, should be an exception. The specialists assure us that diabetes need not necessarily shorten life expectancy if the patient is properly treated. Evidently the majority of diabetics do not enjoy the benefits of modern therapy. The fault is not always the patient's—too often the doctor is amiss. It is evident that both patient and doctor need the same sort of support and help through medical, social, and lay endeavors that have been so successful in the control of other diseases—notably typhoid, tuberculosis, diphtheria, and perhaps appendicitis.

In the hope that something constructive could be done to control heart disease and cancer, national organizations—the American Heart Association and the American Society for the Control of Cancer—were organized. These associations are working in a difficult field, but no one will deny that they have stimulated interest and research and that some progress is being made. It is evident that a national organization for the control of diabetes was needed, and accordingly, there was organized last year the American Diabetes Association, incorporated under the laws of the State of Ohio. Dr. E. P. Joslin is Honorary President, Dr. Herman O. Mosenthal is President, and the membership comprises practically all of the leading specialists in diabetes and physicians interested in diabetes in the country—about 500 in all. The purposes of the American Diabetes Association as outlined in the Constitution are as follows:

ARTICLE I

NAME

This Association shall be a membership corporation not for profit.

ARTICLE II

PURPOSE

1. To disseminate among physicians information relative to the diagnosis and treatment of diabetes by means of meetings, bulletins, publications of papers in scientific journals, and through a central office which would at all times make available information concerning various aspects of diabetes.

2. To educate the laity in the early recognition of diabetes and in the realization of the importance of medical supervision.

3. To secure and co-ordinate the active co-operation of associated groups acceptable to the Trustees in the educational and organizational phases of the Association.

4. To make and publish statistical surveys of diabetes.

5. To encourage and support clinical, experimental, sociological, and statistical studies by means of grants.

6. To encourage the adequate treatment of diabetics and establishment of summer camps for children.

ARTICLE III

MEMBERSHIP

The membership of the Association shall consist of four classes:

1. Active Members
2. Associate Members
3. Honorary Members
4. Corporate Members

If the American Diabetes Association even approximates the success achieved by the National Tuberculosis Association, and there is no reason why it should not, insurance companies that

have become somewhat concerned by the mounting death rate of diabetes will be largely the beneficiaries. In the past, members of the Medical Directors' Association and insurance companies in general have taken a very lukewarm interest in public health, and in the control of diseases causing their heaviest mortalities. Very few insurance companies have lent any encouragement and even fewer any financial support. The excuses offered are either that state insurance commissioners do not favor the expenditure of funds for purposes other than purely insurance, or that the Medical Directors' Association, through its medico-actuarial studies, is adequately contributing to advances in medical sciences. It is not true that state insurance commissioners object to insurance companies supporting national or local health efforts. My Company has, during the past 20 years, made contributions to the national heart and cancer associations, to the American Public Health Association, to our Cincinnati Heart, Cancer, and Diabetes Councils, and even to our Board of Health, with full knowledge of the insurance commissioners.

The second excuse, namely our contributions to medical science from medico-actuarial investigations, might have had some justification some years ago, but as evidenced by our recent programs, our contributions in the future will probably be fewer and less important scientifically. It does not seem entirely fair, or even sporting, that insurance companies should be perfectly willing to reap the harvest and contribute nothing to the labor and expense of sowing and cultivating the crop. This is not solely my personal opinion. Let me quote from a letter that expresses the opinion of many, if not most, of the leading physicians, medical scientists, and those working ardently in the field of public health. The letter was addressed to me and written by Dr. Alan Gregg, 'Director for Medical Sciences' for the Rockefeller Foundation, of which he is also one of the five directors: I quote:

> "One might expect that the businesses chiefly profiting from the improvement in life expectancy (the life

insurance companies) to be generous as well as alert to the source of such favorable gains. Relative to what they and their policyholders have saved as the result of medical research, the life insurance companies' support of medical research deserves no charge of alertness or generosity. There are exceptions and they prove the rule because they are exceptions. But as long as the insurance companies ignore the fact that their relationship to policyholders could be interpreted as contractual and not fiduciary, I expect scant support from the life insurance companies for anything which costs money certainly and only probably will save it many times over later. That is the crucial point: the timidity characteristic of trusteeship."

This rather excoriating criticism is not without justification. It probably expresses the sentiments of the majority of our medical scientists towards us. On the other hand, as in a recent experience I had with the Mayo Clinic, practicing physicians are rather prone to regard every co-operative gesture on the part of insurance companies as actuated by commercial rather than scientific motives. Strangely, in discussing the subject of support for research, etc., with medical directors, I have found that the medical directors of the largest companies are more timorous or reluctant to express any opinion, while those of the smaller companies are frankly in favor—perhaps because most of them are actively in practice and have a clearer viewpoint of problems of medical research and public health.

At any rate, the newly organized American Diabetes Association will be in a position to do much effective work if more adequately financed—and the insurance companies will benefit far beyond the rather meager contribution each one can afford to make. This leaves out of consideration entirely the social and moral obligation for supporting such an association. An annual contribution of $50.00 from every in-

surance company for a corporate membership will enable the American Diabetes Association to carry out fully the purposes of its organization. The Union Central is supporting the Association with an annual contribution of $500.00. I certainly would not recommend to my Company that it do so if I were not thoroughly convinced through my activities since its foundation with the Cincinnati Diabetes Council, that the investment is a sound one.

PRESIDENT CRAGIN—Thank you very much Dr. Muhlberg. You have certainly opened the subject for discussion, if not among the medical directors, at least among our executives at home.

We have a few minutes left before the next feature comes on, and if there is anybody who wants to discuss any of the papers briefly, we will be only too glad to hear from them right now. I wonder if Dr. Alan Gregg won't give us a few words.

DR. GREGG—I think there will be practically no one in the room who will believe me when I say that I didn't know it was at this meeting that Dr. Muhlberg was to quote me, and indeed I didn't know this subject was coming up.

What I wrote him sounded so badly to me that I would like to make it a little clearer as to what I had in mind. My main point was that I don't expect that the life insurance companies are very likely to contribute very much to medical research until it is clearer in the minds of the directors of those companies that their position might be considered as a contractual relationship with the policyholders rather than a fiduciary. To expand that a little bit, contractual relationship virtually says on the death of the insured, the company guarantees to pay so much money, and that is a contract. The fiduciary relationship is, we hold trust funds which are the property, really, of all of our policyholders, and we may not do anything which would not meet with their complete approval and permission.

Let's put this in terms of an imaginary situation to make it a little clearer, an imaginary situation in, say, a family of six children who have inherited a business. They meet in a family council to decide on the question of whether the business will go into a rather large amount of advertising expense, and five members of the family are willing to make an outlay that will have a probable return, an advantage to all, but Sister Emily, who has become a widow and has five children of her own, sees it in the light of an expense which will reduce her immediate and certain income from the family business, and she says, "Thanks very much; no. I have got to have all of the money that I can have out of this family trust for immediate use, and I do not approve of any large expenditure or any expenditure for advertising."

Well, Brother Will says, "Emily, I will make a private agreement with you that will secure to you just as much income as you would have gotten if we didn't go into this advertising business, because I am so sure that if we advertise a little more we will all of us make a lot more money, and so will you."

Now, roughly, my position is that of Brother Will. I think that the life insurance companies, if they put in not necessarily large but some funds in investigative work, would be in a position eventually to profit very considerably, because the record is so clear that they have profited very substantially from medical research work, and I want to make this plea on still one other count. I have a great deal of my time spent looking over requests for grants-in-aid in medical research of a very wide variety. I am very much impressed by the number of perfectly good undertakings which I have to turn down because we haven't the money to do it relative to our other expenditures in other fields, and we haven't the money to do it even absolutely.

What I see happening is a great many of the foundations making a great many small grants-in-aid over short periods of time, one to two to three to, at the outside, five-year

guarantees of sums sometimes as small as $500, sometimes as large as $10,000 a year.

Now, the difficult thing about that from the standpoint of the medical researcher is that he has a three-year goal, we will say, characteristically, at $4,000 a year. He doesn't know where in the devil he will be at the end of that period of time. He, therefore, attacks problems that he is almost sure he can get through with in a short period of time, and so many people have been constrained completely by that kind of consideration, and I would say a little facetiously that about all the small problems available in medical research are being tackled, but there are very few of the long-term things which are being tackled nowadays, and what is needed is this: Combinations or even contributions from single life insurance companies guaranteed over a long period of time that will enable the research men to hire staffs that can look forward to an employment that is not purely precarious, can tackle some of the problems which of their very nature require long-term study, and human life in each instance being a pretty long affair, that is to say, in terms of thirty to fifty to sixty years, develops medical problems which can't be settled in nine academic months of work on guinea pigs or rabbits.

In other words, there are types of medical research which require time for their solution, and I am exceedingly eager to solicit the support of such a character that it can be continued on for long-term undertakings, and I am restless to see that the life insurance companies are at the present time not participating as largely as I think it would profit them to do on two counts; one, the ultimate advantage to their mortality tables, and, two, the good will which I am perfectly certain would be created in the community towards those life insurance companies by that sort of participation, and I say the latter because I think foundations have an amount of good will in the American public quite out of proportion to their actual merits, but quite closely relative to the advantages which their support of medical research has brought to the general public.

PRESIDENT CRAGIN—Thank you very much Dr. Gregg.

We will call on Dr. Dillon to close.

DR. DILLON—The cases which Dr. Root mentioned of insulin resistance are indeed very interesting. Cases of moderate resistance requiring over 100 units of insulin up to 200 units of insulin are quite common affairs. All of us have had a good deal of experience with it. Cases, however, of extreme insulin resistance requiring 500 units and upwards per day are not common. Indeed they are very uncommon. Out of some 7,000 cases in our clinic, we have had only 2, one of them a woman who required 600 to 700 units a day for a long period of time, subsequently went down to no insulin at all, and now after a period of thirteen years of observation, is still living.

Another one was a colored woman who, however, lived only a short time, but I remember one day when we had a visiting doctor in, we demonstrated to him that we could give her 100 units of insulin intravenously and nothing happened to her. I mean by that, she didn't go into any shock or have any reaction of any kind.

These cases, as I say, are very uncommon and very mysterious as to their physiology.

Blood sugar levels I think are very important. There is recent work by Lukens on this particular work, which I believe has not been published, but he did report the work before the meeting of the American Physiological Society last year, in Chicago, and again before the meeting of the Society for Clinical Investigation in Atlantic City last spring. He found that in his animals in which he injected anterior pituitary extract and fluorescin at the same time there was no rise in the blood sugar level, and, therefore, no diabetes developed from the injection of anterior pituitary extract, which would seem to indicate that you cannot produce diabetes in animals experimentally if the blood sugar level is kept down. (You know, of course, that fluorescin causes a large excretion of sugar in the urine so that the blood sugar levels cannot rise.)

All that Dr. Muhlberg and Dr. Gregg have said interests me very much indeed. I think the time has come when we medical directors will have to make up our minds whether we are to remain in the same field of human endeavor that we have been in the past, or whether we are to have a closer liaison with clinical medicine, and particularly with research medicine.

Dr. Muhlberg did not tell you as to how much he personally is responsible for the very excellent work which is being done by the Council on Diabetes in Cincinnati. It is the best public work I know of in any city, and it is largely due to Dr. Muhlberg. Also, Dr. Muhlberg did not tell you that he is the Treasurer of the American Diabetes Association. I tell you these things simply so you will realize to what extent Dr. Muhlberg is taking an interest in medicine outside of the realms of life insurance.

PRESIDENT CRAGIN—Dr. Dillon and your associates, I thank you very much indeed for your very illuminating papers, and it has been a great addition to our program.

I want to announce we have a paper which time did not permit including in the program, but it will be published in the proceedings. The title of the paper is "Comparative Results of Certain Tests for Urinary Sugar," by Dr. Roland A. Behrman, Associate Medical Director and Mr. William T. Wright, A. B., Chief Technician, John Hancock Mutual Life Insurance Company.

Our next feature will be given by Dr. McLeod C. Wilson, of the Travelers Insurance Company, well-known for his wonderful work in the Claim Department. It is a motion picture on "Heart Tolerance Following Coronary Episodes."

. . . Dr. Wilson showed the moving picture . . .

PRESIDENT CRAGIN—I am sure we have all enjoyed Dr. Wilson's presentation.

All good things must come to an end. I have had a very delightful year as President of your Association. I have had

the most co-operative help from everybody. I haven't asked a man for a thing yet but that he has given it to me. Before passing the gavel over to my successor, I just want to express my thanks to you all and my deep appreciation of your kindness.

Dr. Wenstrand, it is your job from now on.

PRESIDENT-ELECT WENSTRAND—Fellow Members of the Association, and Friends: In stepping into this position, I am well aware of the honor that you have conferred upon me as well as my Company, and I wish to thank you most heartily. I also want to thank Dr. Cragin for the very fine program we have had. He has set an example that is going to be hard to beat, but with the help of you all, I hope we will be able to present a program a year from now that will be at least in part as interesting and instructive as the session we have just had.

I can't help but say something about the challenge that Dr. Muhlberg threw out this morning, and Dr. Gregg in his remarks. It gives us something very definite to think about and to shoot at, if I may put it that way. Dr. Root spoke about a bomb; well, perhaps a light bomb has been thrown.

Is there any further business?

DR. JAQUITH—Mr. President, I am sure all who attended the sessions yesterday and today will confirm my belief that this has ranked well with any previous meeting, and I would move that we express to Dr. Cragin our appreciation of the excellent program he has furnished and his conduct of this meeting, and that it be expressed by a rising vote.

(A rising vote of thanks was given Dr. Cragin.)

PRESIDENT-ELECT WENSTRAND—Thank you very much.

Unless there is further business, I will entertain a motion to adjourn this 52nd Annual Meeting of the Association.

* * * * * *

Upon motion, duly seconded and carried, the meeting adjourned.

COMPARATIVE RESULTS OF CERTAIN TESTS FOR URINARY SUGAR

Roland A. Behrman, M. D., Associate Medical Director
William T. Wright, A. B., Chief Technician
John Hancock Mutual Life Insurance Company

The material presented in this paper is concerned with a compilation of tests carried out in the Home Office laboratory of the John Hancock Mutual Life Insurance Company. The study was prompted by the frequency with which questions arose, particularly from field examiners, concerning the significance of certain reactions in various tests for sugar in the urine. We submit therefore the comparative results of several of the standard methods of examination.

Under suitable headings the findings are given in 500 different urines, most of which were selected because they gave an "opaque" Benedict's reaction (this finding is the one most often questioned, although greenish or yellowish reactions with Fehling's solution are equally confusing), but included also are a number of specimens which gave some other reaction to Benedict's test. All tests and readings were performed by one of us (Wright), thus making for uniformity in results and eliminating the confusing factor of several individual opinions as to correct interpretations.

Our present routine in examining for sugar is to test all urines with the picric test and also Fehling's solution.* If picric acid shows 0.2% or more of sugar, the urine is fermented with bakers' yeast for at least an hour for smaller quantities of sugar (up to 1%) and longer for the larger

*The use of Fehling's test is somewhat in the nature of a hold-over from the past when it was the routine and only test, but as it is quite widely used in the field today, it seemed advisable to continue it as a check on the examiner's report. It is, in our experience, a speedy and reliable preliminary method of excluding the possibility of sugar. As we do it, it has never failed to give a suggestive reaction if sugar is present in percentages which we regard as significant, i.e., 0.2% or more. Its chief drawback when depended upon alone is that it may give suggestive or even apparently positive reactions when little or no sugar is present.

quantities at a temperature of about 96°F., and the picric acid test again done on the fermented urine. The difference in percentage readings between the fermented and unfermented urine is, of course, the fermentable—and important—sugar present. No effort is made to identify whatever non-sugar reducing substances may be present. Since there is, to our knowledge, no one direct specific quantitative test for urinary sugar, we believe this expedient is the most reliable for insurance work when dealing with what, as shown by copper tests, seem to be border-line quantities of sugar. All urines showing a reaction of 0.2% or more to the picric test (about 15% of the annual total) are also subjected to the phenylhydrazine and the Benedict's qualitative tests. Details of method in performing the various tests are given at end of paper.

Explanation of our somewhat arbitrary classification of the reactions of the qualitative copper reduction tests now used follows. The refinements in gradation are simply for purposes of comparison with the results of other tests. All readings on copper tests are made immediately upon removal from the hot water bath.

BENEDICT'S QUALITATIVE COPPER TEST

Darker In color only; may be greenish but still clear. Would probably be called negative by all observers.

Very Faint Cloud Usually whitish with no change in color of solution.

Faint Cloud } Greenish tinge, with traces of whitish, or
Cloudy } dull yellor precipitate.

Opaque Always pea green cloud, and after standing gives a trace of dull yellow precipitate.

Heavy Green Denser than opaque.

Yellowish Green
Greenish Yellow
Yellow
Orange
} These terms, we believe, are sufficiently self-descriptive.

FEHLING'S TEST

In addition to the use of any of the foregoing descriptive terms, we add, under the heading of "Fehling's" certain others to describe reactions peculiar to this test:

Clear Brown A clear dark brown associated usually with highly concentrated urine.

Milky Green Opaque light green.

Olive Green Opaque dull green.

Dull Yellow Opaque dull yellow ochre.

Red Opaque brick red.

Brown Opaque dark brown.

PHENYLHYDRAZINE TEST

This test seems to have been regarded as a highly specific one for various types of sugar in the urine, particularly in insurance laboratories of a few years ago, although very few clinicians of our acquaintance are familiar with it; accordingly, a brief discussion may be in order.

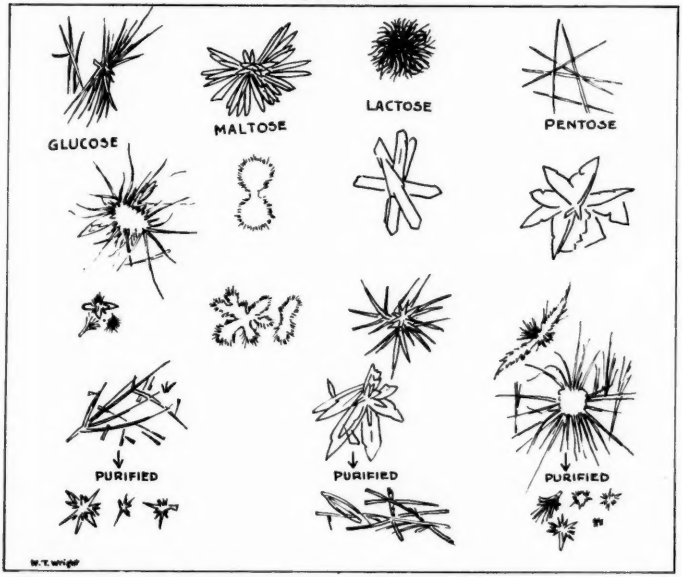
Phenylhydrazine when properly treated reacts with sugar to give characteristic crystals—microscopically identifiable—called osazones. The test, strictly speaking, is a qualitative one only, but the morphology of the osazone crystals varies somewhat according to the parent type of sugar, so that the test is differential in this respect. However, since glucose, pentose, and levulose all yield a similar type of crystal, morphology alone is of limited value in differentiation. Melting-point determinations of the crystals are valuable since the various osazones melt at different temperatures, but purification of the crystals for melting-point determination is a tedious process and not practical in routine work.

It has been pointed out by Everett[1] that uric acid may react with phenylhydrazine, and Dr. Exton[2] has shown that a mixture of sugars will result in unorthodox crystal forms. We have seen a few types which do not conform to the glucose, maltose, lactose, or pentose forms as published in textbooks, and

simply call them "atypical." Everett might call them phenyl-semicarbazide "burrs." Dr. Exton might label them "mixed sugars." We have seen a striking change in the morphology of the same crystals in the process of purification and recrystallization for melting-point determination, so we merely report the test as positive or negative and add that the crystals are "atypical" if they do not conform to the textbook illustrations. We therefore accept the test as a fair qualitative measure but not of particular value for differential purposes.

The accompanying original drawings illustrate various types of crystals.

SOME EXAMPLES OF PUBLISHED AND UNPUBLISHED OSAZONES



Glucose, Maltose, Lactose, Pentose are from illustrations from Hawk. Others are "a-typical" forms, not identified, drawn as seen in course of day's work in John Hancock Laboratory. Bottom row of crystals are same as row above, but purified and recrystallized in preparation for melting point determination. This illustrates how morphology of an osazone may change.

In presenting the tables, the analyses are first grouped according to the reaction to Benedict's qualitative test. The columns, under suitable headings, give the respective findings for that particular test. The specific gravity and albumin content are shown only for possible interest. With the foregoing explanation, it is believed that the heading of each column in the chart and the descriptions set forth thereunder should be sufficiently clear for the reader to draw his own conclusions by comparing the results of the various tests.

From a study of the chart, it is quite evident that most specimens showing 0.2% or more of fermentable sugar give fairly conclusive positive tests by all methods, and particularly striking is the qualitative effectiveness of the Fehling's and phenylhydrazine tests at these percentages. On the other hand, it is equally evident that urines in the opaque green Benedict's group showing less than 0.2% of fermentable sugar do not give uniformly positive, and on the whole not even generally suggestive, sugar reactions to all other methods.

Quoting in part from the original publication of Dr. Benedict's test in *The Journal of the American Medical Association* of October 7, 1911: "In the presence of glucose, the entire body of the solution will be filled with a precipitate which may be red, yellow, or greenish in tinge." The group which we have designated as opaque undoubtedly falls within this interpretation, but we belive our results show quite clearly that an opaque Benedict's test should not be construed as always indicating a significant quantity of sugar. Of 163 urines in this classification, only 23 or 14% contained 0.2% or more of fermentable sugar.

It may be of interest to record that urines were secured from 20 runners at the finish of the 1941 twenty-six mile Boston Marathon Race and 18 of these gave heavy green Benedict's reactions, which would ordinarily have been pronounced positive sugar findings but further examination showed no sugar present.

To illustrate the existing uncertainty of the significance of the finding, we mention that one of the laboratory doctors in a large Boston hospital states that his laboratory records an opaque Benedict's as *negative,* but always requests another specimen. The chief technician in another Boston hospital, where many diabetic cases are treated, states that opaque Benedict's tests are always reported as *positive* for sugar. Quoting from Peters and Van Slyke, "Quantitative Clinical Chemistry", edition of 1937, under the heading "Benign Glycosuria": "Probably thousands of people have been treated for diabetes which they did not have, because their urine gave constantly or intermittently positive reduction tests." This, to be sure, was not written because methods of testing were under question, but we believe that in applying this thought to Selection in Life Insurance or for diagnostic purposes–from a purely practical standpoint–it behooves us to make certain that what may look like a Glycosuria really is one and that such a finding is not due to a false reaction.

REFERENCES

1. Everett, M. R. and Sheppard, F.: The Nature of the Sugar of Normal Urine, J. Biol. Chem. XCVI.431.1932.

2. Exton, W. G.: Differential Diagnosis of Conditions Associated with Sugar Excretion. Read at Annual Meeting N. Y. Med. Soc. 1936.

METHODS

Benedict's Test

9 drops urine
5 cc. Benedict's solution
Boiling water bath 5 minutes
Read immediately upon removal from bath

Fehling's Test

1 Equal quantities of Fehling's A & B freshly mixed.
2 20 drops urine
30 drops Fehling's solution
Place in boiling water bath 2 3/4 minutes.
3. Read immediately upon removal from water bath.

Picric Acid Test

1 cc. urine pipetted into test tube ringed at 25 cc.
3 cc. of 0.2% picric acid solution.
½ cc. of 5% sodium hydroxide.
5 drops 50% acetone, freshly prepared.
Place in boiling water bath 12–14 minutes, cool, add distilled water to mark, mix and read against standards.
For sugar concentration over 0.5% dilute appropriately before testing.

Fermentable Sugar

1. Perform picric acid test on urine.
2. Take about 5 cc. urine in a 15 cc. test tube.
Add about 3 grams fresh yeast—mix.
Incubate at 95°F. for an hour if under 1% sugar—larger quantities overnight.
Centrifuge to clear urine.
3. Subject again to picric acid test.
4. Difference between 1. and 3. is the fermentable sugar.

Phenylhydrazine Test

About 5 cc. urine in 15 cc. test tube.
Add about 1 gram phenylhydrazine hydrochloride crystals.
Add 1–2 grams sodium acetate crystals.
Dissolve, mix, immerse in boiling water bath about 1 hour. Cool slowly.
Examine under microscope for crystals.

COMPARISON OF SEVERAL URINE SUGAR METHODS WITH BENEDICT'S

% FERMENTABLE SUGAR	% PICRIC ACID BEFORE FERMENT	FEHLING'S	PHENYLHYDRAZINE	BENEDICT AFTER FERMENT	SPEC. GRAV.	ALBUMIN
NEGATIVE BENEDICT						
0	0	0	0	0	1005	0
0	0	0	0	0	1005	0
0	0	0	0	0	1007	0.00+
0	0	0	0	0	1008	0
0	0	0	0	0	1016	0.01
0	0.05	0	0	0	1012	0
0	0.10	Darker	0	0	1021	0
0	0.11	0	F.A-typ.	0	1021	0
0	0.20	V.F.Cld.	0	0	1021	0.02
0.01	0.10	0	0	0	1026	0
0.02	0.10	0	F.A-typ.	0	1025	0
0.02	0.10	0	0	0	1025	0.02
0.02	0.12	0	0	0	1026	0
0.03	0.10	0	0	0	1022	0
0.04	0.10	0	F.A-typ.	0	1017	0
0.04	0.11	0	0	0	1025	0
0.04	0.12	0	0	0	1011	0
0.05	0.05	0	0	0	1019	0.01
0.05	0.07	0	0	0	1016	0
0.05	0.08	0	0	0	1018	0.07
0.05	0.09	0	F.A-typ.	0	1024	0
0.05	0.09	0	0	0	1024	0
0.05	0.10	0	0	0	1017	0
0.05	0.13	0	0	0	1026	0
0.06	0.06	0	F.A-typ.	0	1022	0
0.06	0.11	0	0	0	1017	0
0.07	0.09	0	0	0	1026	0
0.07	0.10	0	0	0	1022	0
0.07	0.11	0	F.A-typ.	0	1018	0
0.08	0.08	0	F.A-typ.	0	1006	0
0.08	0.08	0	F.A-typ.	0	1017	0
DARKER BENEDICT						
0	0.06	Darker	0	Darker	1034	0.01
0	0.11	Darker	0	Darker	1022	0.00+
0	0.12	0	0	Darker	1024	0.01
0.01	0.09	Darker	0	Darker	1019	0
0.01	0.20	Cloudy	F.A-typ.	Darker	1028	0
0.01	0.21	Cloudy	F.A-typ.	Darker	1027	0
0.02	0.10	Darker	0	Darker	1026	0
0.03	0.10	0	F.A-typ.	0	1020	0
0.03	0.10	Darker	F.A-typ.	Darker	1031	0
0.03	0.12	0	0	0	1028	0.01
0.07	0.21	Cl.Br.	F.A-typ.	Darker	1019	0.20
VERY FAINT CLOUD BENEDICT						
0	0.06	Darker	0	V.F.Cld.	1023	0.00+
0.01	0.11	0	0	V.F.Cld.	1024	0.00+
0.01	0.20	Darker	F.A-typ.	V.F.Cld.	1025	0
0.02	0.07	0	0	V.F.Cld.	1024	0
0.02	0.11	V.F.Cld.	F.A-typ.	V.F.Cld.	1027	0.19
0.03	0.11	Darker	0	Darker	1028	0
0.03	0.20	V.F.Cld.	0	V.F.Cld.	1024	0.20
0.03	0.20	Darker	0	V.F.Cld.	1031	0.02
0.04	0.10	0	0	V.F.Cld.	1024	0
0.04	0.10	0	F.A-typ.	V.F.Cld.	1026	0.02
0.04	0.14	Darker	0	V.F.Cld.	1026	0
0.04	0.17	Darker	0	0	1030	0
0.05	0.14	Darker	F.A-typ.	0	1026	0
0.05	0.21	Cloudy	0	Cloudy	1033	0
0.07	0.10	0	F.A-typ.	V.F.Cld.	1016	0
0.07	0.12	Darker	0	V.F.Cld.	1027	0.02
0.07	0.20	Darker	0	F.Cld.	1025	0
0.07	0.20	Darker	0	0	1026	0.03
0.08	0.20	Darker	F.A-typ.	V.F.Cld.	1028	0
0.09	0.19	0	F.A-typ.	0	1025	0.02
FAINT CLOUD BENEDICT						
0.01	0.11	Darker	0	V.F.Cld.	1024	0
0.01	0.12	Darker	0	F.Cld.	1026	0
0.02	0.10	Darker	0	V.F.Cld.	1026	0.04
0.02	0.10	0	0	F.Cld.	1027	0.01
0.02	0.13	0	0	V.F.Cld.	1026	0.01
0.02	0.14	Darker	F.A-typ.	V.F.Cld.	1026	0
0.02	0.20	Darker	V.F.Pos.	F.Cld.	1031	0
0.02	0.21	V.F.Cld.	F.A-typ.	V.F.Cld.	1025	0
0.02	0.21	Cloudy	F.A-typ.	F.Cld.	1036	0
0.02	0.24	Opaque	F.Pos.	F.Cld.	1029	0.01
0.02	0.25	Opaque	F.A-typ.	F.Cld.	1035	0.04
0.03	0.20	0	F.A-typ.	F.Cld.	1022	0.00+
0.04	0.14	Darker	F.A-typ.	V.F.Cld.	1031	0
0.04	0.20	V.F.Cld.	0	F.Cld.	1031	0
0.04	0.20	Opaque	0	F.Cld.	1036	0
0.04	0.21	F.Cld.	F.A-typ.	V.F.Cld.	1032	0.01
0.05	0.18	Darker	0	V.F.Cld.	1026	0
0.06	0.11	0	0	V.F.Cld.	1026	0
0.06	0.18	Darker	0	V.F.Cld.	1024	0
0.07	0.20	Cloudy	V.F.Pos.	F.Cld.	1030	0.00+
0.07	0.20	Cl.Br.	F.A-typ.	F.Cld.	1031	0
0.10	0.20	Darker	F.A-typ.	F.Cld.	1030	0
0.11	0.18	0	V.F.Pos.	0	1022	0.03
CLOUDY BENEDICT						
0.02	0.21	Cloudy	F.A-typ.	V.F.Cld.	1024	0.08
0.02	0.23	Cl.Br.	F.A-typ.	Cloudy	1030	0.90
0.03	0.20	Darker	F.A-typ.	Darker	1021	0
0.03	0.20	Opaque	F.A-typ.	Cloudy	1029	0.10
0.03	0.20	V.F.Cld.	V.F.Pos.	F.Cld.	1030	0.01
0.03	0.21	Cloudy	F.A-typ.	F.Cld.	1034	0
0.04	0.13	Darker	0	V.F.Cld.	1023	0.06
0.04	0.14	Darker	0	F.Cld.	1026	0
0.04	0.20	Cloudy	0	F.Cld.	1026	0.01
0.04	0.20	Darker	0	F.Cld.	1032	0.01
0.04	0.22	Cloudy	F.A-typ.	Cloudy	1028	0.01
0.05	0.16	Darker	0	Cloudy	1028	0.22
0.05	0.17	0	0	V.F.Cld.	1025	0
0.05	0.20	Darker	V.F.Pos.	V.F.Cld.	1023	0
0.05	0.20	V.F.Cld.	F.A-typ.	F.Cld.	1026	0
0.05	0.22	Cl.Br.	F.A-typ.	Cloudy	1029	0.00+
0.05	0.22	Darker	V.F.Pos.	F.Cld.	1031	0
0.05	0.25	Cl.Br.	F.A-typ.	Cloudy	1030	0
0.06	0.14	0	0	0	1024	0.01
0.06	0.20	Cloudy	V.F.Pos.	V.F.Cld.	1029	0
0.06	0.23	Cl.Br.	F.A-typ.	Cloudy	1026	0.00+
0.06	0.26	Opaque	0	Cloudy	1030	0
0.07	0.16	V.F.Cld.	0	V.F.Cld.	1022	0
0.07	0.17	Darker	0	V.F.Cld.	1024	0
0.07	0.20	Cloudy	F.Pos.	F.Cld.	1024	0.02
0.07	0.20	Darker	F.A-typ.	V.F.Cld.	1024	0
0.07	0.20	Darker	V.F.Pos.	V.F.Cld.	1030	0
0.07	0.23	Darker	V.F.Pos.	V.F.Cld.	1029	0.01
0.07	0.25	Cloudy	F.Pos.	F.Cld.	1028	0
0.08	0.18	F.Cld.	F.A-typ.	F.Cld.	1026	0
0.08	0.20	F.Cld.	V.F.Pos.	V.F.Cld.	1027	0.01
0.08	0.21	Cloudy	V.F.Pos.	0	1031	0.01
0.08	0.23	F.Cld.	0	V.F.Cld.	1030	0.00+
0.08	0.26	Darker	F.A-typ.	Cloudy	1025	0.50
0.09	0.11	0	V.F.Pos.	0	1016	0
0.09	0.11	Darker	V.F.Pos.	0	1024	0.01
0.09	0.19	V.F.Cld.	F.Pos.	F.Cld.	1020	0.00+

CONTINUATION OF TABLE

% FERMENTABLE SUGAR	% PICRIC ACID BEFORE FERMENT	FEHLING'S	PHENYLHYDRAZINE	BENEDICT AFTER FERMENT	SPEC. GRAV.	ALBUMIN
			CLOUDY BENEDICT (Continued)			
0.09	0.21	F.Cld.	V.F.Pos.	0	1028	0
0.09	0.21	Cloudy	0	F.Cld.	1031	0
0.09	0.26	Opaque	F.Pos.	F.Cld.	1031	0.00+
0.10	0.20	Darker	V.F.Pos.	V.F.Cld.	1020	0.02
0.10	0.25	Opaque	F.A-typ.	Cloudy	1022	0.01
0.11	0.20	Cloudy	V.F.Pos.	F.Cld.	1030	0
0.11	0.21	Cloudy	F.A-typ.	V.F.Cld.	1029	0
0.13	0.27	Cloudy	V.F.Pos.	F.Cld.	1028	0.32
0.14	0.23	Cloudy	V.F.Pos.	F.Cld.	1027	0
0.16	0.27	V.F.Cld.	Pos.	F.Cld.	1023	0.56
0.16	0.32	Cloudy	F.Pos.	Cloudy	1032	0
0.20	0.23	Cloudy	0	0	1024	0
0.23	0.23	0	Pos.	0	1011	0
0.23	0.28	OliveGr.	V.F.Pos.	V.F.Cld.	1020	0
			OPAQUE GREEN BENEDICT			
0.00	0.20	Cloudy	F.A-typ.	Opaque	1032	0
0.00	0.21	V.F.Cld.	F.A-typ.	F.Cld.	1023	0
0.00	0.21	Darker	F.A-typ.	Opaque	1026	0.04
0.00	0.22	V.F.Cld.	0	Opaque	1026	0
0.00	0.24	Cloudy	F.A-typ.	Opaque	1028	0.01
0.00	0.25	Darker	F.A-typ.	Opaque	1020	0
0.00	0.34	Cloudy		Opaque	1028	0.01
0.01	0.20	Cl.Br.	0	Cloudy	1036	0
0.01	0.21	Darker	0	Cloudy	1028	0.03
0.02	0.20	Darker	0	Cloudy	1026	0
0.02	0.20	Darker	F.A-typ.	F.Cld.	1031	0
0.02	0.22	Cloudy	0	V.F.Cld.	1033	0.01
0.02	0.27	Cloudy	V.F.Pos.	Opaque	1034	0.01
0.02	0.30	Opaque	F.A-typ.	Opaque	1034	0.10
0.03	0.20	Cloudy	F.A-typ.	Cloudy	1026	0
0.03	0.20	V.F.Cld.	V.F.Pos.	V.F.Cld.	1030	0
0.03	0.20	Darker	0	Cloudy	1031	0.01
0.03	0.20	V.F.Cld.	F.Pos.	Opaque	1031	0
0.03	0.21	0	0	F.Cld.	1027	0
0.03	0.21	F.Cld.	V.F.Pos.	Opaque	1028	0
0.03	0.22	Cloudy	V.F.Pos.	Cloudy	1028	0.01
0.03	0.23	V.F.Cld.	V.F.Pos.	Opaque	1019	0
0.03	0.23	Darker	0	Cloudy	1029	0
0.03	0.24	DullYell.	F.A-typ.	Cloudy	1020	0.01
0.03	0.27	Cl.Br.	F.A-typ.	Opaque	1031	0
0.03	0.30	Cl.Br.	F.A-typ.	Opaque	1027	0
0.03	0.30	Cl.Br.	F.A-typ.	F.Cld.	1030	0
0.04	0.16	0	0	Opaque	1024	0.02
0.04	0.17	Darker	0	Opaque	1026	0
0.04	0.20	Darker	V.F.Pos.	0	1027	0
0.04	0.20	Darker	F.A-typ.	Opaque	1027	0
0.04	0.20	F.Cld.	V.F.Pos.	V.F.Cld.	1028	0
0.04	0.21	Darker	V.F.Pos.	V.F.Cld.	1029	0
0.04	0.21	Cloudy	F.A-typ.	Opaque	1027	0
0.04	0.21	V.F.Cld.	0	F.Cld.	1030	0
0.04	0.21	F.Cld.	F.Pos.	Cloudy	1032	0.01
0.04	0.26	F.Cld.	F.A-typ.	Opaque	1033	0
0.04	0.27	Cloudy	F.A-typ.	Cloudy	1025	0
0.04	0.27	F.Cld.	V.F.Pos.	Cloudy	1033	0.70
0.04	0.28	V.F.Cld.	0	Opaque	1029	0
0.04	0.30	Cloudy	F.A-typ.	Opaque	1026	0.00+
0.04	0.32	Cloudy	F.A-typ.	Opaque	1022	0
0.05	0.14	0	0	0	1021	0
0.05	0.16	0	0	V.F.Cld.	1017	0.01
0.05	0.20	Opaque	0	Cloudy	1031	0
0.05	0.20	Darker	V.F.Pos.	Cloudy	1029	0
0.05	0.20	Cl.Br.	V.F.Pos.	Opaque	1031	1.10
0.05	0.21	Cloudy	V.F.Pos.	V.F.Cld.	1027	0
0.05	0.21	V.F.Cld.	F.Pos.	V.F.Cld.	1027	0.01
0.05	0.22	Darker	V.F.Pos.	Cloudy	1029	0.00+
0.05	0.22	Cl.Br.	V.F.Pos.	V.F.Cld.	1031	0.09
0.05	0.23	Darker	F.A-typ.	Cloudy	1031	0
0.05	0.33	DullYell.	F.A-typ.	Opaque	1029	0
0.06	0.16	0	0	V.F.Cld.	1023	0.04
0.06	0.17	Darker	V.F.Pos.	Opaque	1026	0
0.06	0.17	Darker	V.F.Pos.	Cloudy	1028	0
0.06	0.20	F.Cld.	F.A-typ.	V.F.Cld.	1027	0
0.06	0.20	V.F.Cld.	0	F.Cld.	1028	0.03
0.06	0.20	Cloudy	V.F.Pos.	Cloudy	1030	0.00+
0.06	0.20	Darker	0	0	1030	0
0.06	0.23	Darker	F.A-typ.	Cloudy	1026	0
0.06	0.23	Darker	F.A-typ.	V.F.Cld.	1033	0.02
0.06	0.24	F.Cld.	0	Opaque	1030	0.04
0.06	0.24	F.Cld.	F.Pos.	F.Cld.	1034	0
0.06	0.28	F.Cld.	V.F.Pos.	Cloudy	1035	0
0.06	0.41	Cl.Br.	F.A-typ.	Opaque	1034	0.10
0.07	0.17	Opaque	F.A-typ.	Cloudy	1016	0
0.07	0.17	Darker	0	V.F.Cld.	1022	0.00+
0.07	0.19	0	F.A-typ.	Cloudy	1026	0
0.07	0.19	Darker	V.F.Pos.	Cloudy	1023	0.01
0.07	0.20	Cloudy	V.F.Pos.	F.Cld.	1020	0
0.07	0.20	Cloudy	0	F.Cld.	1026	0
0.07	0.20	Cloudy	Pos.	Cloudy	1034	0
0.07	0.21	Darker	V.F.Pos.	V.F.Cld.	1016	0
0.07	0.21	Cloudy	0	F.Cld.	1029	0.15
0.07	0.26	Darker	0	Cloudy	1028	0.02
0.07	0.27	V.F.Cld.	F.A-typ.	Opaque	1029	0.03
0.07	0.28	Darker	F.A-typ.	Opaque	1023	0
0.08	0.20	V.F.Cld.	F.Pos.	0	1026	0
0.08	0.20	V.F.Cld.	V.F.Pos.	Opaque	1028	0.01
0.08	0.21	V.F.Cld.	V.F.Pos.	F.Cld.	1031	0
0.08	0.22	Darker	F.A-typ.	F.Cld.	1027	0
0.08	0.25	Cloudy	F.Pos.	F.Cld.	1028	0
0.08	0.26	F.Cld.	V.F.Pos.	Cloudy	1029	0.00+
0.08	0.26	Cloudy	F.Pos.	0	1028	0
0.08	0.28	Opaque	F.A-typ.	Opaque	1029	0.01
0.08	0.29	V.F.Cld.	F.A-typ.	Opaque	1019	0.01
0.09	0.18	Cloudy	F.A-typ.	F.Cld.	1010	0.00+
0.09	0.21	Darker	V.F.Pos.	V.F.Cld.	1027	0
0.09	0.21	Darker	F.Pos.	0	1029	0
0.09	0.26	F.Cld.	F.Pos.	F.Cld.	1025	0.01
0.10	0.20	0	F.Pos.	Cloudy	1014	0
0.10	0.20	Darker	F.Pos.	F.Cld.	1026	0
0.10	0.20	Opaque	0	F.Cld.	1029	0
0.10	0.21	F.Cld.	V.F.Pos.	0	1020	0.00+
0.10	0.21	Darker	F.Pos.	V.F.Cld.	1030	0
0.10	0.27	Orange	Pos.	Cloudy	1016	0
0.11	0.16	0	0	V.F.Cld.	1020	0
0.11	0.17	OliveGr.	V.F.Pos.	0	1012	0.02
0.11	0.20	Darker	V.F.Pos.	V.F.Cld.	1023	0
0.11	0.20	Darker	F.Pos.	F.Cld.	1026	0
0.11	0.22	Opaque	F.Pos.	F.Cld.	1028	0
0.11	0.22	Darker	V.F.Pos.	F.Cld.	1030	0
0.11	0.22	Cloudy	V.F.Pos.	Cloudy	1026	0.00+
0.11	0.24	Cloudy	F.Pos.	F.Cld.	1030	0
0.11	0.35	Cloudy	F.Pos.	Opaque	1030	0
0.12	0.20	Darker	V.F.Pos.	0	1020	0
0.12	0.20	0	V.F.Pos.	0	1024	0
0.12	0.21	Darker	F.Pos.	V.F.Cld.	1027	0
0.12	0.21	Darker	V.F.Pos.	0	1028	0
0.12	0.23	Cloudy	V.F.Pos.	V.F.Cld.	1029	0
0.12	0.24	Darker	F.A-typ.	F.Cld.	1034	0
0.12	0.27	Cloudy	F.Pos.	Cloudy	1031	0.02
0.12	0.37	Opaque	V.F.Pos.	Opaque	1031	0.19
0.12	0.52	OliveGr.	0	Opaque	1034	0.01
0.13	0.20	V.F.Cld.	V.F.Pos.	V.F.Cld.	1028	0
0.13	0.20	V.F.Cld.	V.F.Pos.	V.F.Cld.	1028	0
0.13	0.25	MilkyGr.	Pos.	V.F.Cld.	1020	0
0.13	0.50	Opaque	F.Pos.	Opaque	1033	0.01
0.14	0.19	V.F.Cld.	F.A-typ.	F.Cld.	1027	0

CONTINUATION OF TABLE

OPAQUE GREEN BENEDICT (Continued)

% FERMENTABLE SUGAR	% PICRIC ACID BEFORE FERMENT	FEHLING'S	PHENYLHYDRAZINE	BENEDICT AFTER FERMENT	SPEC. GRAV.	ALBUMIN
0.14	0.26	Cloudy	F.Pos.	0	1018	0.28
0.14	0.31	Darker	V.F.Pos.	F.Cld.	1021	0.00+
0.15	0.20	V.F.Cld.	V.F.Pos.	F.Cld.	1026	0
0.15	0.22	Cloudy	F.Pos.	V.F.Cld.	1026	0
0.16	0.22	Darker	Pos.	V.F.Cld.	1026	0
0.16	0.27	F.Cld.	F.Pos.	V.F.Cld.	1027	0
0.16	0.27	Cloudy	Pos.	V.F.Cld.	1031	0.06
0.16	0.28	Opaque	Pos.	0	1023	0
0.16	0.28	F.Cld.	F.Pos.	F.Cld.	1029	0
0.16	0.33	Cloudy	Pos.	F.Cld.	1032	0
0.17	0.35	Opaque	V.F.Pos.	V.F.Cld.	1032	0
0.18	0.18	0	V.F.Pos.	V.F.Cld.	1016	0
0.18	0.20	Darker	F.Pos.	V.F.Cld.	1019	0
0.18	0.22	V.F.Cld.	V.F.Pos.	V.F.Cld.	1024	0
0.18	0.25	Yell.Gr.	Pos.	V.F.Cld.	1025	0
0.18	0.26	Opaque	Pos.	V.F.Cld.	1030	0
0.18	0.30	OliveGr.	Pos.	V.F.Cld.	1027	0
0.18	0.31	MilkyGr.	Pos.	V.F.Cld.	1029	0
0.19	0.27	Yellow	Pos.	0	1028	0
0.20	0.27	MilkyGr.	V.F.Pos.	0	1026	0
0.20	0.30	MilkyGr.	Pos.	V.F.Cld.	1038	0
0.21	0.26	Opaque	Pos.	0	1025	0.01
0.22	0.32	MilkyGr.	Pos.	F.Cld.	1026	0
0.23	0.23	MilkyGr.	Pos.	0	1016	0
0.23	0.23	V.F.Cld.	F.Pos.	0	1026	0
0.23	0.29	MilkyGr.	Pos.	V.F.Cld.	1021	0
0.23	0.30	MilkyGr.	Pos.	Cloudy	1022	0
0.23	0.32	DullYell.	Pos.	F.Cld.	1029	0
0.25	0.35	OliveGr.	F.Pos.	F.Cld.	1027	0
0.25	0.42	Yellow	Pos.	V.F.Cld.	1025	0
0.26	0.26	Yellow	Pos.	0	1010	0
0.26	0.26	MilkyGr.	Pos.	V.F.Cld.	1025	0
0.26	0.34	Yellow	Pos.	F.Cld.	1021	0.04
0.26	0.38	Yellow	Pos.	F.Cld.	1031	0.00+
0.27	0.27	MilkyGr.	Pos.	F.Cld.	1021	0
0.27	0.27	DullYell.	Pos.	V.F.Cld.	1027	0
0.27	0.35	DullYell.	Pos.	F.Cld.	1024	0.01
0.28	0.38	Green	F.Pos.	Cloudy	1015	0.00+
0.29	0.46	Yellow	Pos.	0	1027	0
0.32	0.32	Yellow	Pos.	V.F.Cld.	1027	0
0.32	0.44	Yellow	Pos.	V.F.Cld.	1023	0.03
0.34	0.34	Yellow	Pos.	0	1029	0
0.50	0.56	Yellow	Pos.	0	1026	0

HEAVY GREEN BENEDICT

0.16	0.28	DullYell.	Pos.	V.F.Cld.	1026	0.02
0.16	0.36	DullYell.	Pos.	Opaque	1028	0
0.17	0.30	Yellow	F.Pos.	Cloudy	1024	0
0.19	0.40	DullYell.	0	Opaque	1035	0
0.20	0.20	Yell.Gr.	Pos.	F.Cld.	1007	0
0.21	0.27	DullGr.	V.F.Pos.	0	1028	0
0.22	0.38	Yellow	F.A-typ.	Opaque	1025	0
0.23	0.33	DullYell.	Pos.	0	1021	0
0.24	0.24	MilkyGr.	Pos.	V.F.Cld.	1015	0
0.25	0.25	Yellow	Pos.	V.F.Cld.	1018	0
0.26	0.38	DullGr.	F.Pos.	Cloudy	1020	0
0.27	0.27	Yellow	Pos.	0	1018	0
0.27	0.40	Yellow	Pos.	V.F.Cld.	1026	0
0.28	0.38	Yellow	Pos.	F.Cld.	1025	0.05
0.30	0.30	Orange	Pos.	0	1015	0.00+
0.30	0.30	Yellow	Pos.	0	1018	0.00+
0.30	0.30	DullYell.	Pos.	V.F.Cld.	1024	0
0.30	0.35	Yellow	Pos.	V.F.Cld.	1022	0.01
0.30	0.36	Yellow	Pos.	V.F.Cld.	1023	0
0.30	0.40	Yellow	Pos.	0	1023	0
0.30	0.42	Yellow	Pos.	F.Cld.	1029	0
0.30	0.48	Yellow	Pos.	Cloudy	1034	0
0.32	0.38	Yellow	Pos.	V.F.Cld.	1026	0
0.32	0.42	Orange	Pos.	V.F.Cld.	1024	0
0.33	0.36	Yellow	Pos.	V.F.Cld.	1020	0.04
0.33	0.44	Yellow	Pos.	F.Cld.	1022	0
0.34	0.44	Yellow	Pos.	0	1027	0
0.35	0.35	DullYell.	Pos.	0	1021	0
0.35	0.40	Yellow	Pos.	0	1018	0
0.36	0.36	DullYell.	Pos.	0	1015	0.02
0.37	0.40	Yellow	Pos.	V.F.Cld.	1021	0
0.37	0.40	Yellow	Pos.	F.Cld.	1027	0
0.38	0.40	Brown	Pos.	0	1004	0
0.40	0.40	Yellow	Pos.	V.F.Cld.	1026	0
0.40	0.46	Orange	Pos.	V.F.Cld.	1026	0.00+
0.40	0.48	Orange	Pos.	F.Cld.	1028	0
0.41	0.50	Yellow	Pos.	0	1026	0
0.41	0.52	DullYell.	Pos.	F.Cld.	1020	0.01
0.42	0.58	Orange	Pos.	F.Cld.	1026	0
0.43	0.57	Yellow	Pos.	F.Cld.	1028	0
0.44	0.46	Orange	V.F.Pos.	0	1025	0.00+
0.46	0.60	Orange	Pos.	F.Cld.	1032	0.04
0.48	0.48	Yellow	V.F.Pos.	0	1027	0
0.49	0.56	Yellow	Pos.	V.F.Cld.	1026	0.00+
0.50	0.56	Orange	Pos.	F.Cld.	1026	0
0.52	0.60	DullYell.	Pos.	V.F.Cld.	1022	0.02
0.61	0.68	Yellow	Pos.	V.F.Cld.	1028	0
3.94	4.14	Brown	Pos.	Opaque	1032	0.02

YELLOW GREEN BENEDICT

0.09	0.54	Yellow	Pos.	Opaque	1026	0.00+
0.20	0.27	DullGr.	Pos.	0	1026	0
0.24	0.42	Yellow	F.A-typ.	V.F.Cld.	1031	0
0.29	0.44	Yellow	Pos.	Cloudy	1025	0.00+
0.34	0.48	Yellow	Pos.	Cloudy	1025	0.02
0.35	0.50	Yellow	Pos.	Cloudy	1032	0
0.37	0.48	Yellow	Pos.	V.F.Cld.	1028	0
0.38	0.46	Orange	Pos.	0	1026	0
0.41	0.58	Yellow	Pos.	Cloudy	1028	0
0.49	0.60	Orange	Pos.	V.F.Cld.	1026	0
0.50	0.50	Orange	Pos.	0	1011	0
0.50	0.50	Orange	Pos.	0	1025	0
0.50	0.54	Orange	Pos.	0	1020	0
0.50	0.60	Yellow	Pos.	0	1036	0
0.54	0.54	Orange	Pos.	0	1016	0
0.56	0.66	Orange	Pos.	V.F.Cld.	1029	0.01
0.57	0.66	Orange	Pos.	0	1020	0.10
0.60	0.60	Yellow	Pos.	V.F.Cld.	1013	0
0.60	0.60	Orange	Pos.	F.Cld.	1017	0
0.60	0.68	Orange	Pos.	0	1023	0
0.61	0.66	Orange	Pos.	V.F.Cld.	1023	0
0.62	0.62	Orange	Pos.	F.Cld.	1023	0
0.64	0.72	Orange	Pos.	0	1028	0
0.69	0.76	Orange	Pos.	F.Cld.	1020	0.01
0.69	0.80	Yellow	Pos.	Cloudy	1026	0
0.75	0.80	Orange	Pos.	F.Cld.	1022	0
0.76	0.80	Orange	Pos.	0	1021	0.00+
0.78	0.95	Orange	Pos.	F.Cld.	1036	0
0.83	0.88	DullYell.	Pos.	0	1024	0.01
0.88	0.88	Orange	Pos.	0	1021	0
0.85	0.90	Brown	Pos.	V.F.Cld.	1027	0
0.90	1.05	Orange	Pos.	V.F.Cld.	1031	0.00+
1.12	1.12	Orange	Pos.	0	1019	0

GREEN YELLOW BENEDICT

0.22	0.40	Yellow	Pos.	Cloudy	1026	0
0.34	0.46	Orange	Pos.	0	1019	0

CONTINUATION OF TABLE

GREEN YELLOW BENEDICT (Continued)

% FERMENTABLE SUGAR	% PICRIC ACID BEFORE FERMENT	FEHLING'S	PHENYLHYDRAZINE	BENEDICT AFTER FERMENT	SPEC. GRAV.	ALBUMIN
0.47	0.62	Orange	Pos.	0	1026	0.01
0.57	0.68	Orange	Pos.	F.Cld.	1030	0.00+
0.55	0.60	Orange	Pos.	0	1031	0
0.60	0.76	Orange	Pos.	F.Cld.	1021	0
0.68	0.68	Orange	Pos.	0	1022	0
0.72	0.72	Orange	Pos.	0	1007	0
0.72	0.72	Orange	Pos.	V.F.Cld.	1011	0.00+
0.72	0.76	Orange	Pos.	F.Cld.	1029	0.03
0.76	0.84	Orange	Pos.	0	1026	0.10
0.80	0.80	Yellow	Pos.	0	1021	0.02
0.85	0.95	Orange	Pos.	F.Cld.	1019	0
0.95	0.95	Orange	Pos.	V.F.Cld.	1026	0
0.95	1.00	Orange	Pos.	F.Cld.	1028	0

YELLOW BENEDICT

0.44	0.52	Yellow	Pos.	V.F.Cld.	1028	0
0.75	0.80	Brown	Pos.	F.Cld.	1027	0
0.80	0.80	Yellow	Pos.	0	1016	0
0.91	1.02	Brown	Pos.	V.F.Cld.	1023	0
0.95	1.00	Brown	Pos.	V.F.Cld.	1019	0
0.96	1.04	Brown	Pos.	F.Cld.	1024	0
1.03	1.10	Brown	Pos.	0	1030	0
1.10	1.20	Brown	Pos.	V.F.Cld.	1031	0
1.14	1.26	Brown	St.Pos.	V.F.Cld.	1030	0
1.15	1.32	Brown	Pos.	0	1037	0
6.50	6.50	Red	St.Pos.	0	1036	0.00+

ORANGE BENEDICT

0.71	0.84	Orange	Pos.	F.Cld.	1027	0.00+
0.80	0.80	Orange	Pos.	V.F.Cld.	1026	0.00+
0.82	0.92	Brown	Pos.	F.Cld.	1031	0.00+
0.90	1.08	Brown	Pos.	V.F.Cld.	1027	0.00+
0.96	0.96	Orange	St.Pos.	0	1021	0
1.00	1.00	Orange	Pos.	0	1013	0.00+
1.08	1.08	Orange	Pos.	V.F.Cld.	1010	0
1.08	1.08	Brown	Pos.	0	1023	0
1.09	1.15	Brown	Pos.	0	1025	0
1.11	1.21	Brown	Pos.	F.Cld.	1028	0
1.16	1.20	Brown	Pos.	0	1021	0
1.20	1.20	Green	Pos.	0	1011	0
1.20	1.20	Brown	Pos.	0	1016	0
1.20	1.20	Brown	Pos.	0	1026	0
1.22	1.26	Brown	Pos.	V.F.Cld.	1024	0
1.23	1.32	Brown	St.Pos.	V.F.Cld.	1026	0.16
1.30	1.44	Brown	St.Pos.	F.Cld.	1035	0.00+
1.32	1.32	Brown	Pos.	V.F.Cld.	1014	0
1.35	1.40	Brown	St.Pos.	V.F.Cld.	1027	0
1.36	1.43	Brown	Pos.	0	1030	0.00+
1.38	1.50	Brown	Pos.	F.Cld.	1032	0
1.40	1.48	Brown	Pos.	V.F.Cld.	1026	0.01
1.40	1.50	Brown	Pos.	0	1029	0.01
1.42	1.50	Brown	Pos.	V.F.Cld.	1025	0
1.50	1.50	Red	St.Pos.	0	1010	0.02
1.54	1.60	Brown	St.Pos.	0	1019	0.10
1.76	1.76	Brown	St.Pos.	0	1017	0
1.77	1.90	Brown	St.Pos.	Opaque	1036	0
1.80	1.90	Brown	St.Pos.	Cloudy	1023	0
1.83	1.90	Brown	St.Pos.	0	1032	0
1.86	2.10	Brown	Pos.	Opaque	1035	0
1.87	1.87	Brown	St.Pos.	F.Cld.	1025	0.00+
1.89	1.89	Brown	Pos.	0	1012	0
1.90	2.00	Brown	Pos.	V.F.Cld.	1034	0
1.92	2.00	Brown	Pos.	F.Cld.	1020	0.00+
1.94	1.98	Brown	Pos.	Darker	1023	0
1.98	1.98	Brown	St.Pos.	0	1021	0.04
2.00	2.00	Brown	St.Pos.	0	1019	0
2.00	2.10	Brown	St.Pos.	F.Cld.	1029	0
2.02	2.10	Brown	St.Pos.	V.F.Cld.	1027	0.02
2.03	2.10	Brown	St.Pos.	0	1026	0.00+
2.09	2.09	Brown	St.Pos.	0	1013	0.00+
2.09	2.09	Brown	St.Pos.	V.F.Cld.	1014	0.01
2.14	2.20	Brown	St.Pos.	V.F.Cld.	1030	0
2.15	2.20	Brown	St.Pos.	0	1024	0
2.15	2.20	Brown	St.Pos.	0	1027	0.01
2.21	2.31	Brown	St.Pos.	V.F.Cld.	1032	0
2.23	2.30	Brown	St.Pos.	0	1025	0.08
2.24	2.24	Brown	St.Pos.	0	1022	0
2.28	2.40	Brown	St.Pos.	F.Cld.	1026	0
2.30	2.42	Brown	Pos.	V.F.Cld.	1029	0
2.52	2.52	Brown	St.Pos.	0	1028	0.02
2.62	2.72	Brown	St.Pos.	F.Cld.	1033	0.01
2.63	2.75	Red	Pos.	F.Cld.	1030	0
2.90	2.97	Brown	St.Pos.	0	1028	0
3.02	3.08	Red	St.Pos.	F.Cld.	1034	0
3.06	3.12	Brown	St.Pos.	0	1026	0.00+
3.06	3.12	Brown	Pos.	0	1027	0
3.07	3.19	Red	St.Pos.	0	1030	0.01
3.08	3.08	Red	St.Pos.	0	1026	0
3.13	3.20	Brown	St.Pos.	0	1020	0
3.13	3.19	Brown	St.Pos.	0	1025	0.05
3.27	3.30	Brown	St.Pos.	V.F.Cld.	1031	0.01
3.35	3.41	Brown	Pos.	V.F.Cld.	1035	0.01
3.36	3.36	Brown	St.Pos.	0	1013	0
3.60	3.63	Brown	St.Pos.	0	1032	0.00+
3.68	3.68	Brown	Pos.	0	1024	0.09
3.93	3.99	Brown	St.Pos.	V.F.Cld.	1030	0
4.07	4.07	Brown	St.Pos.	0	1028	0.00+
4.10	4.20	Brown	St.Pos.	0	1036	0.00+
4.20	4.20	Brown	St.Pos.	0	1025	0.00+
4.29	4.41	Brown	St.Pos.	0	1026	0
4.60	4.60	Brown	Pos.	0	1026	0
4.64	4.64	Brown	St.Pos.	0	1024	0
4.70	4.83	Brown	St.Pos.	F.Cld.	1036	0.01
4.93	5.00	Brown	St.Pos.	0	1029	0.04
4.94	4.94	Brown	Pos.	0	1029	0
5.00	5.00	Brown	St.Pos.	0	1027	0.04
5.07	5.12	Brown	St.Pos.	0	1028	0
5.10	5.10	Brown	St.Pos.	0	1030	0.05
5.20	5.20	Brown	St.Pos.	0	1032	0.00+
5.25	5.25	Brown	St.Pos.	0	1030	0
5.46	5.46	Brown	St.Pos.	0	1036	0.00+
5.80	5.80	Brown	St.Pos.	0	1024	0
5.92	5.92	Brown	St.Pos.	0	1038	0
6.09	6.09	Brown	St.Pos.	Cloudy	1046	0
6.30	6.30	Brown	St.Pos.	0	1029	0
6.70	6.72	Brown	St.Pos.	0	1031	0.02
6.93	6.93	Brown	St.Pos.	0	1033	0
7.25	7.28	Brown	St.Pos.	0	1036	0.00+
7.56	7.56	Brown	St.Pos.	0	1036	0

BROWN BENEDICT

2.77	2.86	Brown	Pos.	0	1031	0
6.60	6.72	Brown	St.Pos.	F.Cld.	1034	0
9.24	9.24	Brown	St.Pos.	0	1034	0

ABBREVIATIONS

F=Faint
Cl=Cloud
Br=Brown
V=Very
Cld=Cloud
Gr=Green
Yell=Yellow
St=Strong
A-typ=A-typical

Members, Delegates and Guests Present 203

The following members, delegates and guests were present at some time during the sessions: Doctors E. V. Allen, G. E. Allen, W. J. Allison, H. H. Amiral, K. W. Anderson, T. D. Archibald, E. M. Armstrong, T. M. Armstrong, W. B. Aten, D. R. Auten, J. A. Avrack, G. H. Barber, N. J. Barker, W. B. Bartlett, E. W. Beckwith, J. E. Bee, R. A. Behrman, M. B. Bender, R. W. Benton, C. C. Berwick, F. P. Bicknell, J. R. Biggs, C. C. Birchard, W. F. Blackford, H. E. Bogart, J. E. Boland, William Bolt, E. C. Bonnett, W. M. Bradshaw, K. F. Brandon, A. O. Bromer, J. L. Brooks, C. T. Brown, F. R. Brown, H. B. Brown, B. C. Bullen, B. F. Byrd, P. E. Carlisle, V. S. Caviness, C. P. Clark, B. R. Comeau, D. B. Cragin, H. W. Crawford, B. B. Crohn, H. C. Cruikshank, R. M. Daley, A. H. Davison, A. C. DeGraff, H. D. Delamere, P. G. Denker, E. J. Dewees, E. G. Dewis, T. H. Dickson, E. S. Dillon, H. W. Dingman, J. P. Donelan, L. I. Dublin, H. L. Dunn, J. T. Eads, O. M. Eakins, H. R. Edwards, J. E. Engelson, J. L. Evans, W. G. Exton, R. K. Farnham, H. H. Fellows, W. E. Ferguson, R. W. Finegan, Frederick Fink, R. A. Fraser, E. M. Freeland, F. I. Ganot, D. S. Garner, J. T. Geiger, W. M. Genthner, E. E. Getman, J. M. Gilchrist, H. E. Goos, R. J. Graves, George Greenway, Alan Gregg, F. L. Grosvenor, R. S. Gubner, J. R. Gudger, R. M. Goepp, Frank Harnden, L. E. Hathaway, J. K. P. Hawks, W. D. Heaton, C. E. Herron, W. L. Hilliard, D. W. Hoare, C. O. Hollinger, J. C. Horan, J. L. Humphreys, J. H. Humphries, J. R. B. Hutchinson, A. S. Irving, C. B. Irwin, W. A. Jaquith, A. O. Jimenis, A. E. Johann, W. M. Jones, H. J. Johnson, G. E. Kanouse, E. A. Keenleyside, H. B. Kennedy, E. F. Kerby, C. E. Kiessling, D. G. Kilgore, C. T. Kirchmaier, H. B. Kirkland, W. P. Lamb, L. H. Lee, E. P. Leeper, G. C. Lockard, H. C. McAlister, C. B. McCulloch, A. J. McGanity, Robert McGrath, George McLean, W. T. McNaughton, F. W. McSorley, Charles Maertz, H. L. Mann, R. W. Mann, W. L. Mann, J. T. Montgomery, J. F. Moore, Jr., William Muhlberg, A. E. Murphy, Eduard Novak, Jan Nyboer, A. J. Oberlander, Herbert Old, M. I. Olsen, A. E. Parks, C. B. Piper, O. S.

Randall, J. H. Ready, P. V. Reinartz, G. P. Robb, D. C. Roberts, A. J. Robinson, H. B. Rollins, H. F. Root, R. L. Rowley, Robert Sanderson, S. B. Scholz, B. T. D. Schwarz, J. O. Segura, J. T. Sheridan, A. L. Sherrill, D. M. Shewbrooks, R. L. Shields, E. E. Simmons, R. R. Simmons, J. L. Siner, W. A. Smith, M. K. Smith, Morton Snow, H. F. Starr, J. B. Steele, D. F. Steuart, E. J. Stieglitz, A. R. Stone, I. R. Stidger, S. J. Streight, E. V. Sweet, B. C. Syverson, L. G. Sykes, H. M. S. Tait, M. B. Thompson, W. E. Thornton, G. D. Townshend, I. G. Towson, Joseph Travenick, Jr., Wallace Troup, H. B. Turner, H. E. Ungerleider, A. P. Upshur, R. C. Voss, W. R. Ward, R. L. Weaver, D. E. W. Wenstrand, C. F. S. Whitney, E. S. Williams, R. L. Willis, M. C. Wilson, S. S. Werth, G. E. Woodford, L. S. Ylvisaker, E. T. Yorke, J. B. Young, and Messrs. L. F. Baker, R. E. Fletcher, Valentine Howell, Arthur Hunter, Edward King, J. J. King, G. C. Kingsley, H. H. Marks, J. A. Mathews, L. N. Parker, George Selser, Pearce Shepherd and P. V. Wells.

Total attendance at all sessions, 207.

MEMORIALS

DR. JOSEPH EGBERT POLLARD

1870–1940

Dr. Pollard died unexpectedly and suddenly at Clearwater, Florida, on November 29, 1940. He had been a member of this Association since 1916 and had been associated with The Prudential since 1909, retiring from active service in 1939.

Born in Newark, Dr. Pollard was a descendant of an old New Jersey family. He attended the Newark public schools and was graduated in medicine from Columbia University in 1894. For four years he was resident surgeon at the Soldiers' Home in Kearny and later served on the surgical staff of St. Michael's Hospital in Newark. In 1900 he entered private practice in Chatham, New Jersey. Nine years later he joined the Medical Department of The Prudential. During the World War, Dr. Pollard served as a captain in the United States Army Medical Corps, being stationed at Fort Totten and the Cunard embarkation pier in New York. When the armistice was signed he was in quarantine at Camp Stewart, Va., preparatory to going overseas.

His associates recognized in him a faithful friend, tested by the close contact of many years. His pleasing personality, good nature, and quiet manner, and his willingness to bear more than his share of responsibilities, marked him as a man of sterling attributes and a dependable friend. His unswerving loyalty under all circumstances was outstanding.

In 1899 he married Mabel A. Davison, who survives him.

DR. OSCAR H. ROGERS

1857–1941

Dr. Oscar H. Rogers, former Chief Medical Director of the New York Life Insurance Company, died of pneumonia at the Massachusetts General Hospital in Boston on May 17, 1941.

He was born on September 6, 1857 in Green Island, New York; son of William C. and Susan Harrison Rogers. He was educated at Troy Academy and graduated from Union College in 1877 with the degree of Civil Engineer. In 1880 he began the study of medicine and graduated in 1883 from the College of Physicians and Surgeons at Columbia University with the degree of Doctor of Medicine.

He became a medical examiner of the New York Life Insurance Company in 1887, and was promoted to the position of Assistant Medical Director in 1894, Medical Director in 1903 and Chief Medical Director in 1912. He held the latter position until he retired from active service on January 1, 1931.

Dr. Rogers was a good mathematician as well as a medical scientist. He made a special study of medical selection from a scientific basis. One of his outstanding achievements was the obtaining of bases of mortality for insuring under-average risks. He secured the necessary information from histories of cases declined for insurance over a period of 15 or more years. The declined cases were classified according to cause of declination; then letters were sent to the applicant, medical examiner, agent and the three friends given in the application. By that means, the records of the dead and of the living were obtained, from which the relative mortality to the normal was found.

Dr. Rogers, in collaboration with the New York Life's actuary, Dr. Arthur Hunter, developed the Numerical System of Ratings, which was soon adopted by many companies in the United States and Canada. A few extracts from the original memorandum of the two authors is given separately.

Dr. Rogers, in collaboration with Dr. Arthur Hunter, was the author of many scientific papers, a few of which may be mentioned as—Graphic Standard Table, The Influence of Occupation Upon Mortality, Numerical Method of Determining the Value of Risks for Life Insurance, Heart Murmurs—Their Influence on Longevity, Blood Pressure as Affected by Sex, Weight, Climate, Altitude, Latitude, or by Abstinence from

Alcoholic Beverages, Past and the Future of Medical Selection, Functional Tests of the Circulation and A Mortality Study of Impaired Lives, under the heading of Inflammatory Rheumatism and Tuberculosis of Lungs and Blood Spitting. Dr. Rogers also contributed to the Mortality Study of Impaired Lives, and was a member of the Medico-Actuarial Committee from 1907 to 1931.

He was Secretary of the Association of Life Insurance Medical Directors from 1894 to 1900, Vice President of the Association from 1904 to 1906, and President of the Association from 1906 to 1908. He was also Chairman of the M. I. B. Committee from 1902 to 1932 and, because of his earnest work on this Committee, he was continued as a Member Emeritus of this Committee until his death.

Dr. Rogers was interested in many inventions, and devised the Dr. Rogers Tycos Sphygmomanometer, an instrument for measuring blood pressure. This instrument is still very largely used by the profession. In 1930 he was awarded an honorary degree of Doctor of Science from Columbia University, and in 1932 he received a similar degree from Union College. In May 1940, he received a "distinguished service citation" for organizing community tuberculosis programs at the annual convention of the State Committee on Tuberculosis and Public Health of the State Charities Aid Association, held in New York.

He was a member of the University Club in New York and for thirteen years was president of the Fortnightly Club for the Study of Anthropology in Yonkers, New York.

Dr. Rogers was filled with compassion for those suffering from physical or mental distress. He was ever ready to serve and help the needy so far as lay within his power. While abhoring cant and hypocrisy in every form, he was a very human and kindly man,—a true friend in need.

He was a great man who will, for many years to come, be greatly missed by his associates in the field of Life Insurance

and by the host of individuals who knew him or knew of his work among us.

The name of Dr. Rogers will always be remembered in connection with the "Numerical Rating System" which was originated jointly by him and Dr. Arthur Hunter. In memory of his passing a few extracts are given of the original, which was entitled "The Scientific Valuation of Lives for Insurance." It was submitted to a Committee of Officers of the New York Life Insurance Company on the 29th of December 1903 and was approved by them.

"MEMORANDUM

re

The Scientific Valuation of Lives for Insurance

In accordance with the instructions of the Executive, Mr. Hunter and Dr. Rogers, acting as a Committee for that purpose, have made a careful study of the data now in the possession of the Company, with a view to devising a plan for more uniform selection of the Company's business, and beg leave to submit the following report.

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SOURCES OF INFORMATION

We have made use of (a) The experience of the Company between 1880 and 1899, inclusive, in the standard class; (b) Of the results of study of the Adjustable Accumulation business brought down to date; (c) Of the results of the work of the Actuarial Society, and (d) Of information from the Reports on Railways of the Interstate Commerce Commission, the Census of 1900, and of information available regarding dangerous trades and occupations.

WHAT IS NEEDED

What is needed is some plan under the operation of which the judgment of the Medical Board in every case may be expressed with accuracy in terms of %M, and to accomplish this it seemed to us that it should be possible to place upon each of the various factors which go to make up a risk a definite valuation which, when added to the values placed upon the other factors, should give us a total expressing the absolute value of the risk. In short,—as the standards thus laid down rest for the greater part upon the judgment of the Medical Board in times past, their summation should be equivalent

to expressing in terms of %M what has been the average judgment of the Medical Board in each type of cases, and what may safely be continued for the future as our standards. * * * * * * *

A plan of this kind worked out to its logical conclusion would produce a more uniform selection throughout the world than we have been able to secure heretofore, because it would enable us to communicate to all of our representatives abroad those minute changes in practice which we are constantly making as our knowledge of medical selection increases.

Up to the present time it has been the practice to instruct members of the Medical Board by word of mouth, and to place definite ratings only on such types of impairment as are clearly sub-standard. In this way, those of the Medical Board who have gone abroad to represent the Medical Department there have carried with them a fairly complete knowledge of the Company's practice up to the time of their departure, but it has been impossible to keep them informed of the minute changes in practice which take place from time to time.

By placing a definite valuation on each factor we may, hereafter, shift these values in accordance with our increased experience, and we may communicate our instructions to each Foreign Office in such form as to be easily understood by the Medical Staff and applied in the current work. Under the operation of such a plan our selection not only will be more clearly defined among the members of our Home Office Medical Board but will be uniform also in the Foreign Departments.

THE PLAN

We decided, therefore, to place a valuation expressed in terms of %M upon each of the factors which make up an insurance risk, namely,—(a) Build, (b) Family History, (c) Habitat, (d) Occupation, (e) Physical Condition, (f) Personal History, (g) Habits, (h) Race.

Of these, the most important is the Build, taken in connection with the Family History, and as it seemed convenient to do so, we included the third factor, Habitat, and we drew up a table expressing the results of our studies of these three combined. * * * * * * *

We placed a valuation on the various degrees of Over and Underweight at the various ages and for persons of "Average" Family History; for cases in which there was one consumptive in the family, two consumptives, three or more consumptives, for persons with pendulous abdomen. We also made allowances for "Deficient" Family History, and also for Family History of "Great Longevity". * * * * * * *

As to the factor of Occupation,—we have made an effort to place on each occupation such valuation as will express the probable influence of that occupation on Longevity, indicating this influence by—Plus 10, Plus 50, Plus 100, or, Minus 10, Minus 15, Minus 30, etc.,—such additions being made to the foundation rating. In the same way we have treated the various other factors, using the plus or minus sign to indicate whether such factor has a favorable or an unfavorable influence, and to what extent.

As to "Physical Condition" and "Personal History", our analysis of the results in our Adjustable Accumulation business has led us to make very few changes,—the Company's experience in most cases being extremely close to the predictions of the Medical Board. In the matter of "Habits" on the other hand, we have been less successful, and a study is now going forward in the Department which will probably enable us to fix some more definite valuations which shall apply to that impairment. * * * * * * *

The ratings submitted by the Committee and which are too bulky to accompany this Report, are tentative, and subject to change from time to time as our experience broadens.

THE TEST

Having outlined a plan and worked out the details as far as possible, it was decided that we should compare the probable results of its application with those now being secured through the present method of selection. Accordingly, we have placed a valuation upon each of 3,000 cases, and have compared our results with those obtained by the Medical Board in the same cases. * * * * * * From this it will be seen that the proposed plan if put in operation would produce a selection somewhat more rigid than that at present in use. It should be noticed, however, that by slightly shifting the standards laid down by the Committee we can raise or lower the selection to just the extent demanded by the needs of the business. * * * * * * *

When these values were being laid down we made an effort to allow about the same margin of safety everywhere, and we therefore feel confident that the procedure we recommend for changing the standard of selection will work out satisfactorily in practice. * * * * * * * *

Respectfully submitted,
ARTHUR HUNTER, Actuary
OSCAR H. ROGERS, Medical Director"

DR. HAROLD F. TAYLOR

1890–1941

Dr. Harold F. Taylor, Associate Medical Director of the Aetna Life Insurance Company died October 22, 1941 of a cerebral hemorrhage in New York, where he had gone to attend a meeting of the Association of Life Insurance Medical Directors as a member of the Committee on Electrocardiographic and X-ray Procedure.

Dr. Taylor was born in Hardwick, Vermont on March 8, 1890 and received his early education there. He was graduated from Dartmouth College in the Class of 1914 with a Bachelor of Science degree. He received his medical degree in 1917 from the University of Vermont.

During the World War he was a First Lieutenant in the United States Army Medical Corps stationed at Fort McPherson in Atlanta, Georgia. On his return to civilian life in 1919 he took up the practice of medicine in Burlington, Vermont and also was an Assistant Professor in Medicine at the University of Vermont.

In 1929 he came with the Aetna Life Insurance Company where his preliminary training, particularly in cardiology, was made full use of, he assuming full charge of all this type of underwriting.

He did much research from the insurance standpoint and his papers and his position on the Cardiac Committee did much to further the knowledge in this field.

Dr. Taylor was a member of Emanual Congregational Church, serving as a deacon for several years, and was prominent in the various activities of the church. He retained his membership in the Vermont Medical Society and was a member of Alpha Kappa Kappa Medical Fraternity and the Wampanoag Country Club.

His wife and three daughters survive him.

Dr. Taylor was well liked by all who met him. He was

an industrious worker and no task was too great for him to tackle. In the last two or three years he had been suffering under considerable handicap but went about his work with the same cheerful spirit which he showed throughout all his life. It is needless to say that he will be greatly missed by his associates and host of friends.

DR. GEORGE RAE WELCH

1902–1941

It is with deep regret that we report the death of Dr. George Rae Welch on May 11, 1941. Dr. Welch's passing at the age of thirty-nine after an illness of only three months, came as a shock to the medical staff of the New York Life Insurance Company and brought with it a feeling of personal loss.

He was born at Clio, South Carolina, in 1902. In 1926 he graduated from the Medical College of South Carolina. After spending four years in the United States Public Health Service he joined the Medical Department of the New York Life Insurance Company in 1930, where at the time of his death he held the position of Medical Supervisor. He was a member of the Life Insurance Medical Directors' Association since 1935.

A man of high ideals, he was loved and respected by his colleagues. He is survived by his widow, one son and one daughter.

DECEASED MEMBERS

John L. Adams, M. D.	New York, N. Y.
Charles D. Alton, M. D.	Hartford, Conn.
Malcolm O. Austin, M. D.	San Francisco, Calif.
Walter C. Bailey, M. D.	Boston, Mass.
Henry A. Baker, M. D.	Kansas City, Mo.
A. W. Barrows, M. D.	Hartford, Conn.
John T. J. Battle, M. D.	Greensboro, N. C.
Wesley W. Beckett, M. D.	Los Angeles, Calif.
Charles D. Bennett, M. D.	Newark, N. J.
Charles Bernacki, M. D.	New York, N. Y.
Thomas W. Bickerton, M. D.	New York, N. Y.
Albert W. Billing, M. D.	New York, N. Y.
Robert J. Blanchard, M. D.	Winnipeg, Man.
Frederick G. Brathwaite, M. D.	New York, N. Y.
William R. Bross, M. D.	New York, N. Y.
Chauncey R. Burr, M. D.	New York, N. Y.
Robert L. Burrage, M. D.	Newark, N. J.
James Campbell, M. D.	Hartford, Conn.
Willard B. Carpenter, M. D.	Columbus, Ohio
Frank W. Chapin, M. D.	New York, N. Y.
Frederick W. Chapin, M. D.	Springfield, Mass.
Ferdinand E. Chatard, M. D.	Baltimore, Md.
Henry Colt, M. D.	Pittsfield, Mass.
Henry W. Cook, M. D.	Minneapolis, Minn.
Thomas C. Craig, M. D.	New York, N. Y.
Edward Curtis, M. D.	New York, N. Y.
Clark W. Davis, M. D.	Cincinnati, Ohio
William B. Davis, M. D.	Cincinnati, Ohio
Charles A. Devendorf, M. D.	Detroit, Mich.
Henry K. Dillard, M. D.	Philadelphia, Pa.
Frank Donaldson, M. D.	Baltimore, Md.
Percy G. Drake, M. D.	Hartford, Conn.
Edwin W. Dwight, M. D.	Boston, Mass.
James B. Eagleson, M. D.	Seattle, Wash.
Z. Taylor Emery, M. D.	New York, N. Y.
John W. Fisher, M. D.	Milwaukee, Wis.

DECEASED MEMBERS

Paul FitzGerald, M. D.	Newark, N. J.
Thomas A. Foster, M. D.	Portland, Me.
Samuel W. Gadd, M. D.	Philadelphia, Pa.
Homer Gage, M. D.	Worcester, Mass.
Thomas H. Gage, M. D.	Worcester, Mass.
Donald M. Gedge, M. D.	New York, N. Y.
Walter R. Gillette, M. D.	New York, N. Y.
Frank S. Grant, M. D.	New York, N. Y.
Frederick L. Grasett, M. D.	Toronto, Can.
Landon Carter Gray, M. D.	New York, N. Y.
Frederick W. Hagney, M. D.	Newark, N. J.
Ignatius Haines, M. D.	Boston, Mass.
George C. Hall, M. D.	Richmond, Va.
Joseph B. Hall, M. D.	Hartford, Conn.
Edward H. Hamill, M. D.	Newark, N. J.
William W. Hitchcock, M. D.	Los Angeles, Calif.
Angier B. Hobbs, M. D.	New York, N. Y.
Donald C. Hoffman, M. D.	New York, N. Y.
Edgar Holden, M. D.	Newark, N. J.
John Homans, M. D.	Boston, Mass.
John Homans, 2d, M. D.	Boston, Mass.
J. Charles Humphreys, M. D.	Philadelphia, Pa.
Abel Huntington, M. D.	New York, N. Y.
Ross Huston, M. D.	Des Moines, Iowa
Henry H. Hutchison, M. D.	Toronto, Can.
Lefferts Hutton, M. D.	New York, N. Y.
Phineas H. Ingalls, M. D.	Hartford, Conn.
Arthur Jukes Johnson, M. D.	Toronto, Ont.
John M. Keating, M. D.	Philadelphia, Pa.
Edward B. Kellogg, M. D.	Boston, Mass.
William W. Knight, M. D.	Hartford, Conn.
Edward Lambert, M. D.	New York, N. Y.
John B. Lewis, M. D.	Hartford, Conn.
Ernest H. Lines, M. D.	New York, N. Y.
John M. Little, M. D.	Boston, Mass.
Robert L. Lounsberry, M. D.	Binghamton, N. Y.

DECEASED MEMBERS

Henry P. Lyster, M. D.	Detroit, Mich.
Milton T. McCarty, M. D.	Frankfort, Ind.
Charles N. McCloud, M. D.	St. Paul, Minn.
Francis A. McGreen, M. D.	New York, N. Y.
Lewis McKnight, M. D.	Milwaukee, Wis.
Thomas H. McMahon, M. D.	Toronto, Ont.
Elias J. Marsh, M. D.	Paterson, N. J.
Henry A. Martelle, M. D.	Hartford, Conn.
Allison Maxwell, M. D.	Indianapolis, Ind.
Archibald Mercer, M. D.	Newark, N. J.
Francis D. Merchant, M. D.	New York, N. Y.
William R. Miller, M. D.	Hartford, Conn.
William D. Morgan, M. D.	Hartford, Conn.
John P. Munn, M. D.	New York, N. Y.
William Natress, M. D.	Toronto, Ont.
Charles T. Necker, M. D.	Waterloo, Can.
Edwin M. Northcott, M. D.	Portland, Me.
Brace W. Paddock, M. D.	Pittsfield, Mass.
Frank K. Paddock, M. D.	Pittsfield, Mass.
William A. Peterson, M. D.	Chicago, Ill.
Joseph E. Pollard, M. D.	Newark, N. J.
William E. Porter, M. D.	New York, N. Y.
Albert T. Post, M. D.	New York, N. Y.
James T. Priestly, M. D.	Des Moines, Iowa
William W. Quinlan, M. D.	Chicago, Ill.
Oliver P. Rex, M. D.	Philadelphia, Pa.
Thomas H. Rockwell, M. D.	New York, N. Y.
Oscar H. Rogers, M. D.	New York, N. Y.
Edward K. Root, M. D.	Hartford, Conn.
James F. W. Ross, M. D.	Toronto, Ont.
Charles L. Rudasill, M. D.	Richmond, Va.
Gurdon W. Russell, M. D.	Hartford, Conn.
George R. Shepherd, M. D.	Hartford, Conn.
Donald W. Skeel, M. D.	Los Angeles, Calif.
Dewitt Smith, M. D.	Dallas, Tex.
George S. Stebbins, M. D.	Springfield, Mass.
George S. Strathy, M. D.	Toronto, Ont.
Melancthon Storrs, M. D.	Hartford, Conn.

DECEASED MEMBERS

Brandreth Symonds, M. D.	New York, N. Y.
H. Cabell Tabb, M. D.	Richmond, Va.
Harold F. Taylor, M. D.	Hartford, Conn.
James Thorburn, M. D.	Toronto, Ont.
James D. Thorburn, M. D.	Toronto, Ont.
William Thorndike, M. D.	Milwaukee, Wis.
Paul E. Tiemann, M. D.	New York, N. Y.
Harry Toulmin, M. D.	Philadelphia, Pa.
Frank L. Truitt, M. D.	Indianapolis, Ind.
Henry Tuck, M. D.	New York, N. Y.
John S. Turner, M. D.	Dallas, Tex.
Joseph P. Turner, M. D.	Greensboro, N. C.
S. Oakley Van der Poel, M. D.	New York, N. Y.
Charles A. VanDervoort, M. D.	Philadelphia, Pa.
A. L. Vanderwater, M. D.	New York, N. Y.
Clinton D. W. VanDyck, M. D.	New York, N. Y.
George A. VanWagenen, M. D.	Newark, N. J.
Aaron C. Ward, M. D.	Newark, N. J.
William Perry Watson, M. D.	Newark, N. J.
Joseph H. Webb, M. D.	Waterloo, Ont.
William E. H. Wehner, M. D.	Philadelphia, Pa.
Faneuil S. Weisse, M. D.	New York, N. Y.
George R. Welch, M. D.	New York, N. Y.
Ernest A. Wells, M. D.	Hartford, Conn.
Frank Wells, M. D.	Boston, Mass.
Franklin C. Wells, M. D.	New York, N. Y.
George W. Wells, M. D.	New York, N. Y.
Charles D. Wheeler, M. D.	Worcester, Mass.
A. H. Whitridge, M. D.	Baltimore, Md.
George Wilkins, M. D.	Montreal, Que.
Thomas H. Willard, M. D.	New York, N. Y.
Gordon Wilson, M. D.	Baltimore, Md.
G. S. Winston, M. D.	New York, N. Y.
Harry P. Woley, M. D.	New York, N. Y.
Albert Wood, M. D.	Worcester, Mass.
Green V. Woolen, M. D.	Indianapolis, Ind.
John C. Young, M. D.	Detroit, Mich.
Joseph C. Young, M. D.	Newark, N. J.

LIST OF MEMBERS OF THE ASSOCIATION OF LIFE INSURANCE MEDICAL DIRECTORS

George E. Allen, M. D.	National Life, Montpelier, Vt.
Wilfred J. Allison, M. D.	Southwestern Life, Dallas, Tex.
Hiram H. Amiral, M. D.	State Mutual Life, Worcester, Mass.
Henry H. Amsden, M. D.	United Life & Accident, Concord, N. H.
Karl W. Anderson, M. D.	Northwestern National, Minneapolis, Minn.
Harry B. Anderson, M. D.	Imperial Life, Toronto, Canada.
Robert B. Andrews, M. D.	Massachusetts Mutual, Springfield, Mass.
Thomas D. Archibald, M. D.	North American, Toronto, Canada.
Edward M. Armstrong, M. D.	Mutual Life, New York City.
Thomas M. Armstrong, M. D.	Philadelphia Life, Philadelphia, Pa.
William B. Aten, M. D.	Security Mutual, Binghamton, N. Y.
J. Albert Avrack, M. D.	United States Life, New York City.
Henry A. Bancel, M. D.	Mutual Life, New York City.
G. Holbrook Barber, M. D.	Manhattan Life, New York City.
Norman J. Barker, M. D.	Connecticut General, Hartford, Conn.
William B. Bartlett, M. D.	John Hancock, Boston, Mass.
Edgar W. Beckwith, M. D.	Equitable Life Assurance, New York City.
James E. Bee, M. D.	Kansas City Life, Kansas City, Mo.
Roland A. Behrman, M. D.	John Hancock, Boston, Mass.

Maurice B. Bender, M. D.	Guardian Life, New York City.
Roy W. Benton, M. D.	Northwestern Mutual, Milwaukee, Wis.
Charles C. Berwick, M. D.	Metropolitan Life, New York City.
Francis P. Bicknell, M. D.	State Mutual Life, Worcester, Mass.
Edward B. Bigelow, M. D.	State Mutual Life, Worcester, Mass.
Cecil C. Birchard, M. D.	Sun Life, Montreal, Canada.
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Philadelphia Life Insurance Co., Philadelphia, Pa.	T. M. Armstrong, M. D.

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Pilot Life Insurance Co., Greensboro, N. C.	H. F. Starr, M. D.
The Praetorians, Dallas, Texas.	E. P. Leeper, M. D.
Prov. Life & Acci. Ins. Co., Chattanooga, Tenn.	C. R. Henry, M. D.
Provident Mutual Life Ins. Co., Philadelphia, Pa.	E. J. Dewees, M. D. Herbert Old, M. D.
Prudential Insurance Co., Newark, N. J.	C. T. Brown, M. D. P. E. Carlisle, M. D. E. G. Dewis, M. D. W. G. Exton, M. D. F. I. Ganot, M. D. W. C. Hausheer, M. D. G. E. Kanouse, M. D. H. B. Kirkland, M. D. W. P. Lamb, M. D. P. V. Reinartz, M. D. M. K. Smith, M. D. I. R. Stidger, M. D. L. S. Ylvisaker, M. D. E. T. Yorke, M. D.
Reliance Life Insurance Co., Pittsburgh, Pa.	O. M. Eakins, M. D. Kenneth Gardner, M. D. W. W. Hobson, M. D. J. L. Humphreys, M. D.
Reserve Loan Life Insurance Co., Dallas, Tex.	S. A. Shelburne, M. D.
Security Life & Accident, Denver, Colo.	C. H. Willis, M. D.
Security Mutual Life Ins. Co., Binghamton, N. Y.	W. B. Aten, M. D.
Shenandoah Life Insurance Co., Roanoke, Va.	D. S. Garner, M. D.

Southland Life Insurance Co., Dallas, Tex.	J. T. Montgomery, M. D. Hall Shannon, M. D.
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State Farm Life Ins. Co., Bloomington, Ill.	J. K. P. Hawks, M. D.
State Life Insurance Co., Indianapolis, Ind.	F. T. Hallam, M. D. C. B. McCulloch, M. D.
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Sun Life Assurance Co., Montreal, Canada.	C. C. Birchard, M. D. J. K. Gordon, M. D. A. W. Young, M. D.
Travelers Insurance Company, Hartford, Conn.	J. T. Cabaniss, M. D. W. W. Dinsmore, M. D. R. M. Filson, M. D. L. C. Grau, M. D. F. L. Grosvenor, M. D. G. H. Shaw, M. D. Euen Van Kleeck, M. D. M. C. Wilson, M. D.
Union Central Life Ins. Co., Cincinnati, Ohio.	Charles Maertz, M. D. William Muhlberg, M. D. W. O. Pauli, M. D.
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United States Life Ins. Co., New York City.	J. A. Avrack, M. D.
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West Coast Life Insurance Co., San Francisco, Calif.	I. C. Heron, M. D.
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Woodmen of the World Life Ins. Society, Omaha, Neb.	A. D. Cloyd, M. D. H. B. Kennedy, M. D.

Author Index

VOL. XXVIII—1941

Note—Titles of Original Papers in Plain Type;
of Papers Discussed, in *Italics*

Page

Allen, Edgar V. Chronic Occlusive Peripheral Arterial Diseases .. 141

Behrman, Roland A. Comparative Results of Certain Tests for Urinary Sugar 192

Bolt, William. *Prognosis and Insurability of Hypertension with Particular Reference to the Electrocardiogram* 40

Clark, Charles P. *The Transverse Diameter of the Frontal Aortic Silhouette* 65

Crohn, Burrill B. Achlorhydria: Its Ultimate Significance 74

Daley, Robert M. Prognosis and Insurability of Hypertension with Particular Reference to the Electrocardiogram 18

Dillon, Edward S. Recent Developments in Diabetes 157

Dublin, Louis I. The Inheritance of Longevity—A Study Based Upon Life Insurance Records 89

Dunn, Halbert L. Vital Statistics and Life Insurance in a Changing World 128

Fellows, Haynes H. *The Transverse Diameter of the Frontal Aortic Silhouette* 61

Gregg, Allen. *Recent Developments in Diabetes* 186

Gubner, Richard S. Prognosis and Insurability of Hypertension with Particular Reference to the Electrocardiogram 18

Marks, Herbert H. The Inheritance of Longevity—A Study Based Upon Life Insurance Records 89

Muhlberg, William. *Recent Developments in Diabetes* 178

Root, Howard F. *Recent Developments in Diabetes* 169

Sheridan, Joyce T. The Transverse Diameter of the Frontal Aortic Silhouette 49

Stieglitz, Edward J. *The Inheritance of Longevity—A Study Based Upon Life Insurance Records* 121

Ungerleider, Harry E. Prognosis and Insurability of Hypertension with Particular Reference to the Electrocardiogram 18

Wilson, McLeod C. Heart Tolerance Following Coronary Episodes —a motion picture 190

Wright, William T. Comparative Results of Certain Tests for Urinary Sugar 192

Subject Index

VOL. XXVIII—1941

Note— Names of Authors in Plain Type;
of Discussers, in *Italics*.

Page

Address: Presidential, Cragin 1
Chairman: Medical Section, American Life Convention, Blackford 4
Edwards, Herbert R., New York City Board of Health .. 6

Achlorhydria: Its Ultimate Significance, Crohn 74

Aortic Silhouette, The Transverse Diameter of the Frontal, Sheridan. *Fellows, Clark* 49, 61, 65

Cumulative Author Index, 1937-1941 243

Cumulative Subject Index, 1937–1941 249

Diabetes, Recent Developments in, Dillon. *Root, Muhlberg, Gregg* 157, 169, 178, 186

Heart Tolerance Following Coronary Episodes,—a motion picture shown by Wilson 190

Hypertension with Particular Reference to the Electrocardiogram, Prognosis and Insurability of, Daley, Ungerleider, Gubner. *Bolt* 18, 40

Longevity—A Study Based upon Life Insurance Records, The Inheritance of, Dublin, Marks. *Stieglitz* 89, 121

Members of Association:
Active, 217; Companies and Representatives, 231; Deceased, 213; Emeritus, 230; Honorary, 229.

Obituaries:
Pollard, Joseph Egbert, 205; Rogers, Oscar H., 205; Taylor, Harold F., 211; Welch, George Rae, 212.

Peripheral Arterial Diseases, Chronic Occlusive, Allen 141

Transverse Diameter of the Frontal Aortic Silhouette, The, Sheridan. *Fellows, Clark* 49, 61, 65

Urinary Sugar, Comparative Results of Certain Tests for, Behrman, Wright 192

Vital Statistics and Life Insurance in a Changing World, Dunn 128

Cumulative Author Index

1937—1941

Note—Titles of Original Papers in Plain Type; of Papers Discussed, in *Italics*

	Volume	Page
Allen, Edgar V. Chronic Occlusive Peripheral Arterial Diseases	XXVIII	141
Allison, Wilfred J. *Notes on One Method of Underwriting Electrocardiograms*	XXVI	138
Disorders Due to Nutritional Deficiency	XXVII	64
Anderson, H. B. Poliomyelitis and Wind-borne Pollens	XXIV	86
Anderson, Karl W. *A Study of the Transverse Diameter of the Heart Silhouette with Prediction Table Based on the Teleoroentgenogram*	XXV	102
Baehr, George. The Relative Role of the Kidneys and of the Blood Vessels in the Hypertensive Cardiovascular Renal Diseases	XXVI	75
Beckwith, E. W., Brathwaite, Mills. Glycosuria: Mortality	XXIV	8
Behrman, Roland A., Wright. Comparative Results of Certain Tests for Urinary Sugar	XXVIII	192
Bell, E. T. Primary Hypertension: A Clinical and Pathological Study of 1,520 Cases with Especial Reference to Renal Arteriosclerosis	XXVI	269
Bender, M. B., Chairman, Medical Section, American Life Convention	XXVI	22
Birchard, C. C. *Coronary Disease and Life Insurance*	XXVI	231
Birchard, C. C., Gordon. Diabetes and Inheritance	XXIV	92
Blackford, W. E., Chairman, Medical Section, American Life Convention	XXVIII	4
Bolt, William. *Past Hypertension and Present Average Pressure*	XXIV	172
Prognosis and Insurability of Hypertension with Particular Reference to the Electrocardiogram	XXVIII	40
Bonnett, Earl C. Notes on One Method of Underwriting Electrocardiograms	XXVI	120
Bowman, J. A. Thornley. *Anemias, Hemorrhagic Disorders*	XXV	277
Bradshaw, William M. *Glomerulonephritis, The Modern Conception of*	XXV	129
Brathwaite, F. G., Beckwith, Mills. Glycosuria: Mortality	XXIV	8

Cumulative Author Index–cont.

	Volume	Page
Brown, C. T. Presidential Address	XXIV	5
Carber, F. H. Goiter: Fifteen Years' Experience	XXIV	212
Christiernin, C. L. Gerontology—The National Institute of Health of the United States Public Health Service	XXVII	11
Christiernin, C. L., Dublin, Marks. A Mortality Study of Applicants for Insurance Showing Albuminuria and Casts	XXVI	160
Clark, Charles P. *The Transverse Diameter of the Frontal Aortic Silhouette*	XXVIII	65
Clark, Charles P., Ungerleider. A Study of the Transverse Diameter of the Heart Silhouette with Prediction Table Based on the Teleoroentgenogram	XXV	84
Cook, H. W. *Rheumatic Heart Disease*	XXV	192
Presidential Address	XXVI	3
Cragin, Donald B. Presidential Address	XXVIII	1
Cragin, Donald B., Roberts. Detection of Metallic Poisons and Some Types of Anemia. A Preliminary Report	XXVII	18
Crawford, J. Hamilton. *A Study of the Transverse Diameter of the Heart Silhouette with Prediction Table Based on the Teleoroentgenogram*	XXV	95
Crile, George. Indications for Operation and End-Results of the Treatment of Essential Hypertension by Celiac Ganglionectomy	XXVI	17
Crohn, Burrill B. Achlorhydria: Its Ultimate Significance	XXVIII	74
Daley, Robert M. Brain Tumors: *Clinical Prognosis and Underwriting Experiences*	XXVI	254
Daley, Robert M., Ungerleider, Gubner. Prognosis and Insurability of Hypertension with Particular Reference to the Electrocardiogram	XXVIII	18
DeGraff, A. C. *Electrocardiography: Recent Advances*	XXIV	154
de la Chapelle, Clarence C. *A Study of the Transverse Diameter of the Heart Silhouette with Prediction Table Based on the Teleoroentgenogram*	XXV	98
Denker, P. G. Psychoneuroses. Prognosis, Expectancy	XXIV	179
Brain Tumors: Clinical Prognosis and Underwriting Experiences	XXVI	237
Dillon, Edward S. *Glycosuria: Mortality*	XXIV	38
Recent Developments in Diabetes	XXVIII	157
Don Carlos, H. S., Wilson. Disability Claim Handling	XXV	30
Downing, John Godwin. *Serology—A Problem*	XXVII	38

Cumulative Author Index–cont.

	Volume	Page
Dublin, Louis I., Marks. Women: Mortality by Build ...	XXIV	47
Women: Mortality by Build	XXV	203
The Inheritance of Longevity—A Study Based Upon Life Insurance Records	XXVIII	89
Dublin, Louis I., Christiernin, Marks. A Mortality Study of Applicants for Insurance Showing Albuminuria and Casts	XXVI	160
Dunn, Halbert L. Vital Statistics and Life Insurance in a Changing World	XXVIII	128
Eakins, O. M. Queer? Or are We?	XXV	155
Edwards, Herbert R.—New York City Board of Health. Address	XXVIII	6
Exton, William G. *Serology—A Problem*	XXVII	41
Fahr, George E. Glomerulonephritis, The Modern Conception of	XXV	104
Fellows, Haynes H. *The Transverse Diameter of the Frontal Aortic Silhouette*	XXVIII	61
Ferguson, W. E. *Past Hypertension and Present Average Pressure*	XXIV	175
Forkner, Claude E. Anemias, Hemorrhagic Disorders ..	XXV	258
Frost, Harold M. *Women: Mortality by Build*	XXV	226
Presidential Address	XXVII	13
Goodell, R. A. *Coronary Disease*	XXVI	109
Gordon, J. K., Birchard. Diabetes and Inheritance	XXIV	92
Gregg, Allen. *Recent Developments in Diabetes*	XXVIII	186
Gubner, Richard S., Daley, Ungerleider. Prognosis and Insurability of Hypertension with Particular Reference to the Electrocardiogram	XXVIII	18
Gudger, James R., Willis. Coronary Disease	XXVI	94
Hall, Llewellyn. *Glycosuria: .Mortality*	XXIV	29
Hedley, O. F. Rheumatic Heart Disease	XXV	163
Herron, Carroll E. *Disorders Due to Nutritional Deficiency*	XXVII	57
Hoare, Daniel W. *Disability Claim Handling*	XXV	69
Hunter, Arthur. Remarks, Actuarial and Medical Problems	XXV	232
Irving, J. Grant. *The Present Status of Chemotherapy in the Treatment of Infections*	XXVII	110
Johann, A. E., Chairman, Medical Section, American Life Convention	XXV	8

Cumulative Author Index–cont.

	Volume	Page
Johnson, Harry J., Short. Glucose Tolerance	XXV	237
Johnson, Harry J. *Coronary Disease*	XXVI	114
Joslin, Elliott P. *Glucose Tolerance*	XXV	252
Keefer, Chester S. The Present Status of Chemotherapy in the Treatment of Infections	XXVII	85
Kennedy, Foster. *Psychoneuroses: Prognosis, Expectancy*	XXIV	195
King, Jos. E. J. *Brain Tumors: Clinical Prognosis and Underwriting Experiences*	XXVI	252
Kirkland, Henry B. *Glomerulonephritis, The Modern Conception of*	XXV	126
The Two Kinds of Asthma	XXVII	77
Kountz, W. B. Revival of Human Heart (Film)	XXIV	95
Lahey, Frank H., President-Elect, American Medical Association	XXVII	3
Le Moine, Marguerite, Rein, Stephens. Serology—A Problem	XXVII	26
Livingston, J. M. *Past Hypertension and Present Average Pressure*	XXIV	168
MacKenzie, L. F., Shepherd. Past Hypertension and Present Average Pressure	XXIV	157
Marks, Herbert H., Dublin. Women: Mortality by Build	XXIV	47
Women: Mortality by Build	XXV	203
The Inheritance of Longevity—A Study Based Upon Life Insurance Records	XXVIII	89
Marks, Herbert H., Dublin, Christiernin. A Mortality Study of Applicants for Insurance Showing Albuminuria and Casts	XXVI	160
McCreight, George. *Goiter: Fifteen Years' Experience*	XXIV	221
McCrudden, F. H. *Glycosuria: Mortality*	XXIV	34
McMillan, Thomas M. *Rheumatic Heart Disease*	XXV	186
Means, James H. Endocrine Disorders in Relation to Insurability	XXVII	159
Metcalf, R. K. *Disability Claim Handling*	XXV	79
Mills, J. P., Brathwaite, Beckwith. Glycosuria: Mortality	XXIV	8
Minot, George R. (Simmons, R. R.) *Anemias and Hemorrhagic Disorder*	XXV	279
Moore, Merrill. Alcoholism: A Public Health Problem	XXVII	127
Moorhead, John J. Trauma and Disability	XXV	60

Cumulative Author Index–cont.

	Volume	Page
Muhlberg, William. *Queer? Or Are We?*	XXV	159
Coronary Disease and Life Insurance	XXVI	225
Recent Developments in Diabetes	XXVIII	178
Murphy, Ray D. Selecting Large Risks, Recent Advances in	XXV	133
Medical and Actuarial Co-operation During the Past Fifty Years	XXVI	9
Myrick, Julian. President, National Association Life Underwriters	XXVI	23
Neustatter, Otto. Address of Greeting	XXIV	45
Old, Herbert. *Disorders Due to Nutritional Deficiency*	XXVII	65
Pauli, W. O. *Goiter: Fifteen Years Experience*	XXIV	217
Rackemann, Francis M. The Two Kinds of Asthma	XXVII	69
Rein, Charles R., LeMoine, Stephens. Serology—A Problem	XXVII	26
Roberts, Sidney H., Cragin. Detection of Metallic Poisons and Some Types of Anemia—A Preliminary Report	XXVII	18
Robinson, A. J. Chairman, Medical Section, American Life Convention	XXVII	12
Root, Howard F. *Recent Developments in Diabetes*	XXVIII	169
Rowntree, L. G. *A Mortality Study of Applicants for Insurance Showing Albuminuria and Casts*	XXVI	191
Scholz, Samuel B. Presidential Address	XXV	3
Shapiro, H. L. *Women: Mortality by Build*	XXIV	77
Shepherd, Pearce. *Women: Mortality by Build*	XXV	223
Juvenile Insurance	XXVI	58
Shepherd, Pearce, MacKenzie. Past Hypertension and Present Average Pressure	XXIV	157
Sheridan, Joyce T. The Transverse Diameter of the Frontal Aortic Silhouette	XXVIII	49
Shewbrooks, Daniel M. *Endocrine Disorders in Relation to Insurability*	XXVII	172
Short, James J., Johnson. Glucose Tolerance	XXV	237
Smith, George Willard. President, New England Mutual Life Insurance Company	XXVII	8
Steele, John B. *Disorders Due to Nutritional Deficiency*	XXVII	63
Stephens, Margaret, Rein, Le Moine. Serology—A Problem	XXVII	26

Cumulative Author Index–cont.

	Volume	Page
Stieglitz, Edward J. *The Inheritance of Longevity—A Study Based Upon Life Insurance Records*	XXVIII	121
Strauss, Maurice B. Disorders Due to Nutritional Deficiency	XXVII	46
Streight, S. J. *Juvenile Insurance*	XXVI	64
Alcoholism: A Public Health Problem	XXVII	156
Thornton, Walter E. Address: Chairman, Medical Section, American Life Convention	XXIV	3
Juvenile Insurance	XXVI	29
Alcoholism: A Public Health Problem	XXVII	143
Ungerleider, Harry E. *Revival of Human Heart (Film)*	XXIV	95
Notes on One Method of Underwriting Electrocardiograms	XXVI	155
Ungerleider, Harry E., Clark. A Study of the Transverse Diameter of the Heart Silhouette with Prediction Table Based on the Teleoroentgenogram	XXV	84
Ungerleider, Harry E., Daley, Gubner. Prognosis and Insurability of Hypertension with Particular Reference to the Electrocardiogram	XXVIII	18
Wallace, Charles K. *Serology—A Problem*	XXVII	40
Willis, R. L. *Women: Mortality by Build*	XXIV	81
Willis, R. L., Gudger. Coronary Disease	XXVI	94
Willius, Fred A. Coronary Disease and Life Insurance	XXVI	215
Wilson, F. N. Electrocardiography: Recent Advances	XXIV	96
Wilson, McLeod C. Heart Tolerance Following Coronary Episodes—a motion picture	XXVIII	190
Wilson, McLeod C., Don Carlos. Disability Claim Handling	XXV	30
Wright, William T., Behrman. Comparative Results of Certain Tests for Urinary Sugar	XXVIII	192
Ylvisaker, Lauritz. *Notes on One Method of Underwriting Electrocardiograms*	XXVI	144

Cumulative Subject Index

1937 — 1941

Note—The Names of Authors are Printed in Plain Type; of Discussers, in *Italics.*

	Volume	Page
Achlorhydria: Its Ultimate Significance. Crohn	XXVIII	74
Actuarial and Medical Problems, Remarks. Hunter	XXV	232
Actuarial Co-operation During the Past Fifty Years, Medical and. Murphy	XXVI	9
Addresses: Chairmen, Medical Section, American Life Convention. Thornton, XXIV, 3; Johann, XXV, 8; Bender, XXVI, 22, Robinson, XXVII, 12; Blackford, XXVIII, 4.		
Addresses: Other Neustatter, XXIV, 45; Myrick, XXVI, 23; Lahey, XXVII, 3; Smith, XXVII, 8; Edwards, XXVIII, 6.		
Addresses: Presidential Brown, XXIV, 5; Scholz, XXV, 3; Cook, XXVI, 3; Frost, XXVII, 13; Cragin, XXVIII, 1.		
Albuminuria and Casts: A Mortality Study of Applicants for Insurance Showing. Christiernin, Dublin, Marks. *Rowntree*	XXVI	160
Alcoholism: A Public Health Problem. Moore. *Thornton, Streight*	XXVII	127
Anemia, Detection of Metallic Poisons and Some Types of—A Preliminary Report. Cragin, Roberts	XXVII	18
Anemias, Hemorrhagic Disorders. Forkner. *Bowman, Minot (Simmons, R. R.)*	XXV	258
Aortic Silhouette, The Transverse Diameter of the Frontal. Sheridan. *Fellows, Clark*	XXVIII	49, 61, 65
Asthma, The Two Kinds of. Rackemann. *Kirkland*	XXVII	69
Blood Pressure: (See "Hypertension") Preliminary Notes and Report of Committee on. Scholz	XXVI	256
Blood Sugar: Glucose Tolerance. Johnson, Short. *Joslin*	XXV	237
Brain Tumors: Clinical Prognosis and Underwriting Experiences. Denker. *Daley, King*	XXVI	237
Build: Women: Mortality by Build. Dublin, Marks. *Shapiro, Willis*	XXIV	47
Women: Mortality by Build. Dublin, Marks. *Frost, Shepherd*	XXV	203

Cumulative Subject Index–cont.

	Volume	Page
Cardiovascular Renal Diseases, The Relative Role of the Kidneys and of the Blood Vessels in the Hypertensive. Baehr	XXVI	75
Casts: A Mortality Study of Applicants for Insurance Showing, Albuminuria and. Christiernin, Dublin, Marks. *Rowntree*	XXVI	160
Celiac Ganglionectomy, Indications for Operation and End-Results of the Treatment of Essential Hypertension by. Crile	XXVI	17
Chemotherapy in the Treatment of Infections, The Present Status of. Keefer. *Irving*	XXVII	85
Coronary Disease. Gudger, Willis. *Goodell, Johnson*	XXVI	94
Coronary Disease and Life Insurance. Willius. *Muhlberg, Birchard*	XXVI	215
Death Certificates, A Report of the Committee on	XXV	10
Diabetes and Inheritance. Gordon, Birchard	XXIV	92
Diabetes, Recent Developments in. Dillon. *Root, Muhlberg, Gregg*	XXVIII	157, 169, 178, 186
Disability Claim Handling. Don Carlos, Wilson. *Hoare, Metcalf*	XXV	30
Disability, Trauma and Moorhead	XXV	60
Electrocardiogram, Prognosis and Insurability of Hypertension with Particular Reference to the. Daley, Ungerleider, Gubner. *Bolt*	XXVIII	18, 40
Electrocardiograms, Notes on One Method of Underwriting. Bonnett. *Allison, Ylvisaker, Ungerleider*	XXVI	120
Electrocardiography: Recent Advances. Wilson. *DeGraff*	XXIV	96
Endocrine Disorders in Relation to Insurability. Means. *Shewbrooks*	XXVII	159
Gerontology. Christiernin	XXVII	11
Glomerulonephritis, The Modern Conception of. Fahr. *Bradshaw, Kirkland*	XXV	104
Glucose Tolerance. Johnson, Short. *Joslin*	XXV	237
Glycosuria: Further Mortality Study. Brathwaite, Beckwith, Mills. *Hall, McCrudden, Dillon*	XXIV	8
Goiter: Fifteen Years' Experience. Carber. *Pauli, McCreight*	XXIV	212

Cumulative Subject Index–cont.

	Volume	Page
Heart:		
Heart: Human, Revival of (Film). Kountz. *Ungerleider*	XXIV	95
Rheumatic Heart Disease. Hedley. *Cook, McMillan* ..	XXV	163
Heart Tolerance Following Coronary Episodes—a motion picture—Wilson	XXVIII	190
A Study of the Transverse Diameter of the Heart Silhouette with Prediction Table Based on the Teleoroentgenogram. Clark, Ungerleider. *Anderson, Crawford, de la Chapelle*	XXV	84
Hypertension: Past, and Present Average Pressure. MacKenzie, Shepherd. *Livingston, Bolt, Ferguson* ..	XXIV	157
Hypertension by Celiac Ganglionectomy, Indications for Operation and End-Results of the Treatment of Essential. Crile	XXVI	17
Hypertension with Particular Reference to the Electrocardiogram, Prognosis and Insurability of. Daley, Ungerleider, Gubner. *Bolt*	XXVIII	18, 40
Infantile Paralysis:		
Poliomyelitis and Wind-borne Pollens. Anderson	XXIV	86
Infections, The Present Status of Chemotherapy in the Treatment of. Keefer. *Irving*	XXVII	85
Inheritance, Diabetes and. Gordon, Birchard	XXIV	92
Inheritance of Longevity—A Study Based upon Life Insurance Records, The. Dublin, Marks. *Stieglitz*	XXVIII	89, 121
Juvenile Insurance. Thornton. *Shepherd, Streight*	XXVI	29
Life Insurance:		
Coronary Disease and Life Insurance. Willius. *Muhlberg, Birchard*	XXVI	215
Juvenile Insurance. Thornton. *Shepherd, Streight* ...	XXVI	29
Recent Advances in Selecting Large Risks. Murphy ..	XXV	133
Vital Statistics and Life Insurance in a Changing World. Dunn	XXVIII	128
Longevity—A Study Based upon Life Insurance Records, The Inheritance of. Dublin, Marks. *Stieglitz*	XXVIII	89, 121
Medical and Actuarial Co-operation During the Past Fifty Years. Murphy	XXVI	9
Medical Problems, Actuarial and—Remarks. Hunter	XXV	232
Members of the Association:		
Active. XXIV, 238; XXV, 300; XXVI, 313; XXVII, 197; XXVIII, 217.		
Companies and Representatives. XXIV, 251; XXV, 313; XXVI, 326; XXVII, 210; XXVIII, 231.		
Deceased. (See "Obituaries"). XXIV, 235; XXV, 296; XXVI, 309; XXVII, 193; XXVIII, 205.		
Emeritus. XXIV, 250; XXV, 312; XXVI, 325; XXVII, 209; XXVIII, 230.		
Honorary. XXIV, 250; XXV, 312; XXVI, 325; XXVII, 209; XXVIII, 229.		

Cumulative Subject Index–cont.

	Volume	Page
Metallic Poisons and Some Types of Anemia, Detection of—A preliminary Report. Cragin, Roberts	XXVII	18
Mortality:		
Albuminuria and Casts: A Mortality Study of Applicants for Insurance Showing. Christiernin, Dublin, Marks. *Rowntree*	XXVI	160
Glycosuria: Further Mortality Study. Brathwaite, Beckwith, Mills. *Hall, McCrudden, Dillon*	XXIV	8
Women: Mortality by Build. Dublin, Marks. *Shapiro, Willis*	XXIV	47
Women: Mortality by Build. Dublin, Marks. *Frost, Shepherd*	XXV	203
Motion Pictures:		
Revival of Human Heart, Kountz. *Ungerleider*	XXIV	95
Heart Tolerance Following Coronary Episodes. Wilson	XXVIII	190
Nutritional Deficiency, Disorders Due to. Strauss. *Herron, Steele, Allison, Old*	XXVII	46
Obituaries: (See "Members Deceased")		
Alton, Charles D.	XXIV	229
Austin, Malcolm O.	XXVI	299
Baker, Henry Anthony	XXVII	183
Bailey, Walter Channing	XXV	287
Battle, John Thomas Johnson	XXVII	184
Brathwaite, Frederick G.	XXVI	301
Cook, Henry Wireman	XXVII	185
Drake, Percy Greenough	XXVI	302
Fisher, John Welton	XXVI	303
Gage, Homer	XXV	288
Hagney, Frederick W.	XXVI	306
Hobbs, Angier Bailey	XXV	289
Hoffman, Donald C.	XXV	290
Huston, Ross	XXV	291
McCarty, Milton T.	XXV	292
Morgan, William D.	XXIV	230
Necker, C. T.	XXVI	307
Pollard, Joseph Egbert	XXVIII	205
Quinlan, William Wheelock	XXV	293
Rockwell, Thomas H.	XXIV	231
Rogers, Oscar H.	XXVIII	205
Root, Edward K.	XXIV	232
Rudasill, Charles Louis	XXV	293
Skeel, Donald W.	XXVII	187
Smith, DeWitt	XXIV	233
Taylor, Harold F.	XXVIII	211
Van Wagenen, George Alexander	XXVII	189
Weisse, Faneuil Suydam	XXVII	191
Welch, George Rae	XXVIII	212
Wheeler, Charles D.	XXV	294
Peripheral Arterial Diseases, Chronic Occlusive. Allen	XXVIII	141

Cumulative Subject Index–cont.

	Volume	Page
Poliomyelitis and Wind-borne Pollens. Anderson	XXIV	86
Psychoneuroses: Prognosis, Expectancy. Denker. *Kennedy*	XXIV	179
Public Health Problem, Alcoholism: A. Moore. *Thornton, Streight*	XXVII	127
Public Health: Address. Edwards	XXVIII	6
Queer? Or Are We? Eakins. *Muhlberg*	XXV	155
Rheumatic Heart Disease. Hedley. *Cook, McMillan* ...	XXV	163
Selecting Large Risks, Recent Advances in. Murphy	XXV	133
Serology—A Problem. Rein, Stephens, LeMoine. *Downing, Wallace, Exton*	XXVII	26
Teleoroentgenogram, A Study of the Transverse Diameter of the Heart Silhouette with Prediction Table Based on the. Clark, Ungerleider. *Anderson, Crawford, de la Chapelle*	XXV	84
Transverse Diameter of the Frontal Aortic Silhouette, The. Sheridan. *Fellows, Clark*	XXVIII	49, 61, 65
Transverse Diameter of the Heart Silhouette with Prediction Table Based on the Teleoroentgenogram, A Study of the. Clark, Ungerleider. *Anderson, Crawford, de la Chapelle*	XXV	84
Trauma, Disability. Moorhead	XXV	60
Tumors, Brain: Clinical Prognosis and Underwriting Experiences. Denker. *Daley, King*	XXVI	237
Urinary Sugar, Comparative Results of Certain Tests for. Behrman, Wright	XXVIII	192
Vital Statistics and Life Insurance in a Changing World. Dunn	XXVIII	128
Women: Mortality by Build. Dublin, Marks. *Shapiro, Willis*	XXIV	47
Women: Mortality by Build. Dublin, Marks. *Frost, Shepherd*	XXV	203